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OF THE

THIRTY-FOURTH ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

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ATLANTIC CITY, N. J.

OCTOBER 21-25, 1929

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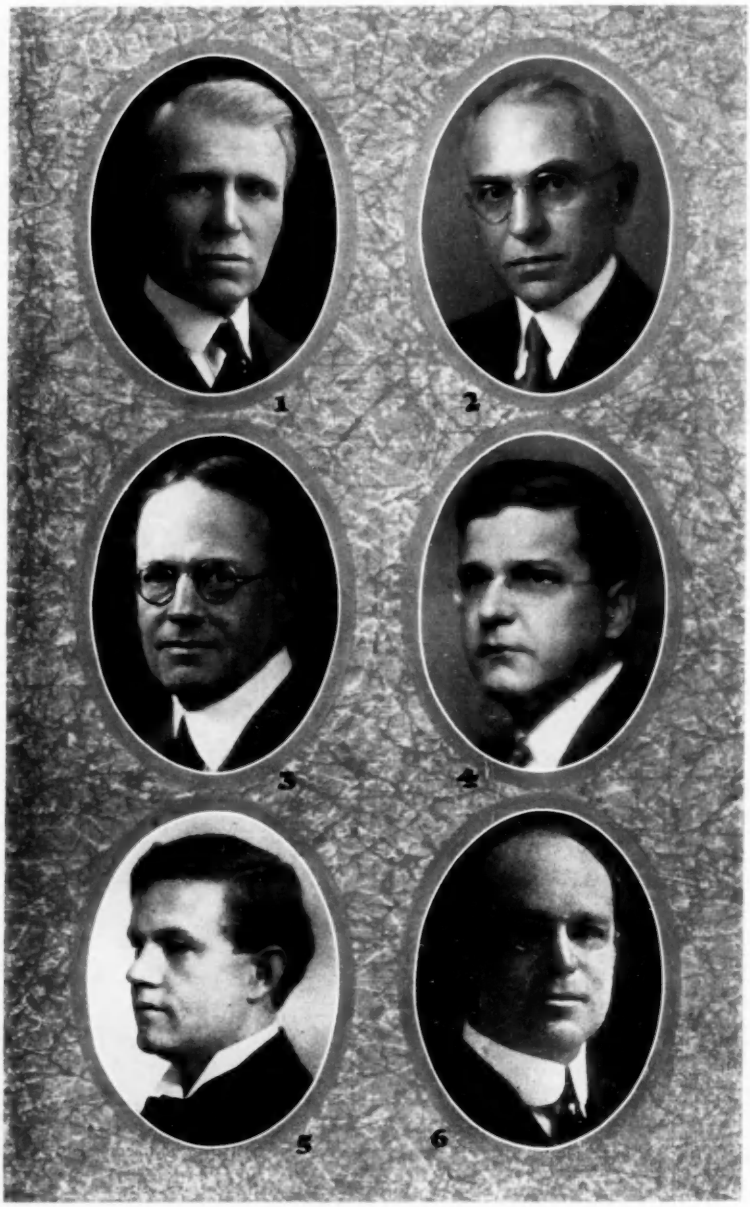
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GOLF

Two trophy cups are now in possession of the Society. One was donated by the St. Louis Fellows of the Academy in 1928, and the other, herewith pictured, is the gift of The Hotel Traymore, Atlantic City, headquarters of the 1929 meeting.

JOINT SESSION

PRESIDENT'S ADDRESS

THE SUBMAXILLARY FOSSA APPROACH TO DEEP PUS IN THE NECK

HARRIS P. MOSHER, M. D.

BOSTON, MASS.

This is not an address; it is a short paper on the applied anatomy of the side (chiefly the upper half) of the neck. I am aware that it skirts the elementary at times, but it is the elementary, or the fundamental, which stands by in emergency. Those who side-step the surgery of the neck will be bored by it. The paper is based on my operating room experience, and on dead-house study. It gives a conception of the neck which has often steadied my hand and reassured me in this serious work.

THE PURPOSE

Pus in the neck calls for the surgeon's best judgment, his best skill, and often for all of his courage. Pus in the neck generally kills by oedema of the larynx, or by the burrowing pus bursting its bounds and flooding the larynx and trachea. Such cases are soon over. There is, however, a more lingering type of infection in the neck. In these cases there may have been frank pus, generally about the tonsil. This is soon evacuated by surgery or by nature, but the patient sinks into a long drawn out septicemia and dies. In such cases there is a thrombosis of the internal jugular vein comparable in every way to thrombosis of the lateral sinus. The latter condition is well known to the profession and all surgeons are on the watch for it. Thrombosis of the internal jugular vein following infection in and about the tonsil is not so well recognized. Years ago I thought, in the pride of my youth, that I had cleared the air by a paper on this subject. American literature is seldom read beyond this country except by one or two of the Allies. A paper on this subject by a member of the staff of an European clinic—and a splendid one by the way—was given before this society within a year or two, pitched in the

ringing tones of a discoverer. Spleen aside, what I want to say is this: **thrombosis of the internal jugular vein from tonsil infection still takes its weekly toll under the guise of septicemia of unknown origin. Washington, as you will remember,** died from oedema of the larynx following a throat infection.

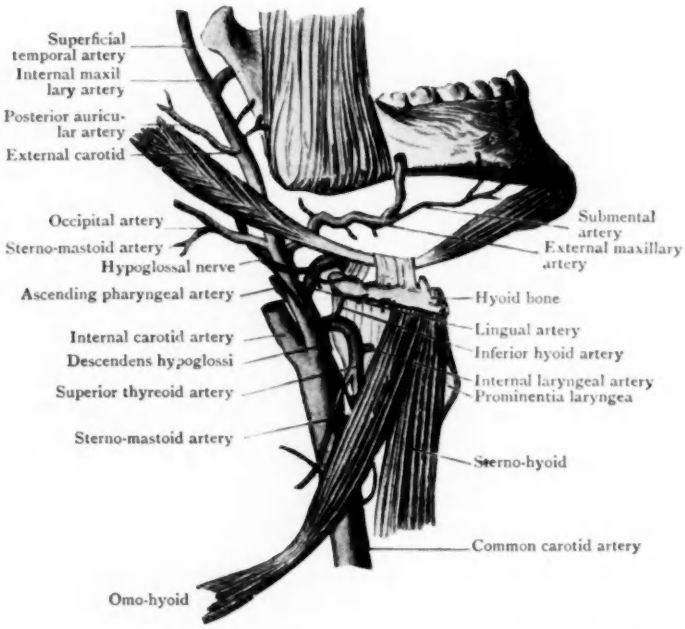


Diagram of the External Carotid Artery and its Branches. The right half of the mandible is tilted up.

(Cunningham)

FIG. 1

The surgeries are full of landmarks for operations on the neck. For practical purposes there are too many. The bony landmarks which have served me best are the following: in the middle line, the cricoid cartilage; on the side of the neck the tip of the great horn of the hyoid bone; high in the neck and deep, the styloid process.

The muscular landmarks are: below the hyoid bone the inner edge of the sterno cleido mastoid muscle; above the hyoid bone the posterior belly of the digastric muscle. All of us have known almost from the cradle, that the great vessels of the neck are found under the inner edge of the sterno mastoid muscle. It took me a long time, however, to sense the fact that the posterior belly of the digastric muscle is an equally sure guide

POSTERIOR BELLY OF DIGASTRIC MUSCLE
WHARTON'S DUCT AND DEEP PART OF GLAND
HYPOGLOSSAL NERVE
INTERNAL JUGULAR
GREAT CORNU OF HYOID
LINGUAL TRIANGLE, WITH COURSE OF LING. ARTERY COVERED BY HYO-GLOSSUS

Submaxillary triangle and the deep relations of the submaxillary gland. Dotted white line indicates the position of the gland, dotted red line the course of the lingual artery beneath the hyoglossus. (Testut.)

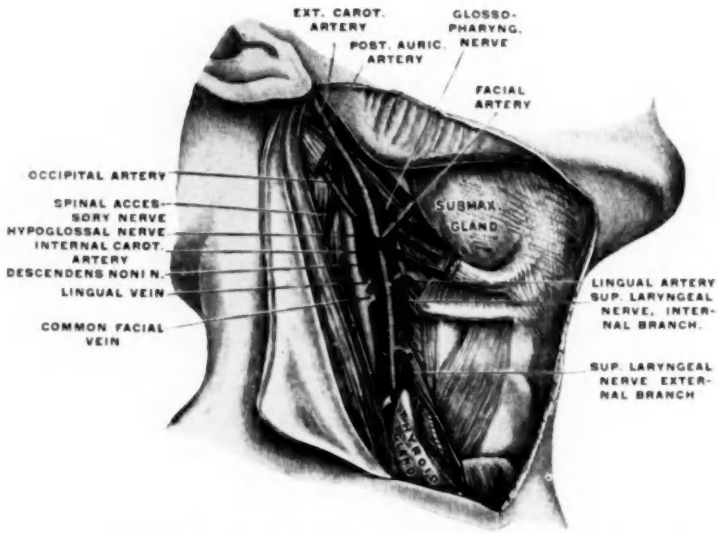
FIG. 2

to the great vessels high in the neck. They lie just behind it. It would be an insult to you for me to feel that I must describe the carotid sheath, its relations and contents; yet applicants before the American Board often fail on this fundamental question, regardless of the fact that I ask it year after year.

The most superficial of the major soft structures of the neck is the hypoglossal nerve. The operator should be on the watch for it almost as soon as he has finished his skin incision.

The Cervical Fasciae

The cervical fasciae are both very real and very unreal. They are always very unreal on an examination paper. Admitting, of course, that they can be divided as they are in the anatomies there is a much simpler and more practical way of con-



Structures of the upper part of neck in the superior carotid and submaxillary triangles. The sternomastoid muscle is retracted somewhat backward, the digastric is divided and the omohyoid removed. (Zuckerkandl.)

FIG. 3

sidering them. It is anatomical heresy, I am aware, to simplify the cervical fasciae as I have done. Students, however, hail it. The purpose of the fasciae, as I see it, is to keep everything in the neck snugly in its alloted place. Without them swan-like necks would be impossible. To me the fasciae are but prolonga-

tions or offshoots of the carotid sheath. My advice is to remember as many of their names as you conveniently can but to consider them in this practical way. This conception of the carotid sheath and the cervical fasciae makes the sheath the core of the soft structures of the neck. Such in surgery it is. I like to call it the Lincoln Highway. By following it you reach the base of the skull above, the chest below. Pus does the same thing. It may accumulate about the sheath almost out of reach at the base of the skull. In the side of the neck it may collect along the sheath or in the sheath. All of this spells to me the important applied point that once the operator reaches the carotid sheath he can follow it to the base of the skull or in the reverse direction to the thorax. Pus anywhere deep in the side of the neck cannot be missed by such an exploration. In other words, in hunting for pus deep in the neck find the Lincoln Highway—that is, the carotid sheath—and follow it. It is the natural highway for pus and for the surgeon in pursuit of pus.

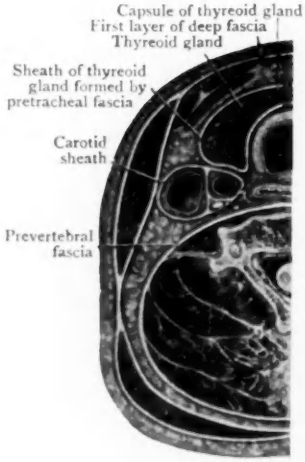


Diagram of deep Cervical Fascia in transverse section at the level of the thyreoid gland.

(Cunningham)

FIG. 4

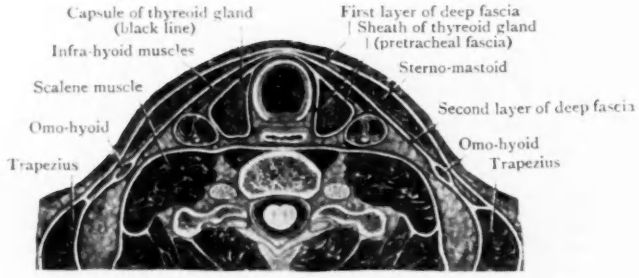


Diagram of the deep Cervical Fascia in a transverse section of the lower part of the neck.

(Cunningham)

FIG. 5

From the standpoint of the fasciae and the muscles the hyoid bone divides the front of the neck into two parts. The muscles of the tongue run down to it. The muscles which cover the trachea and the thyroid gland run upward from the sternum and are attached to it. The first layer of the deep fasciae—the split-

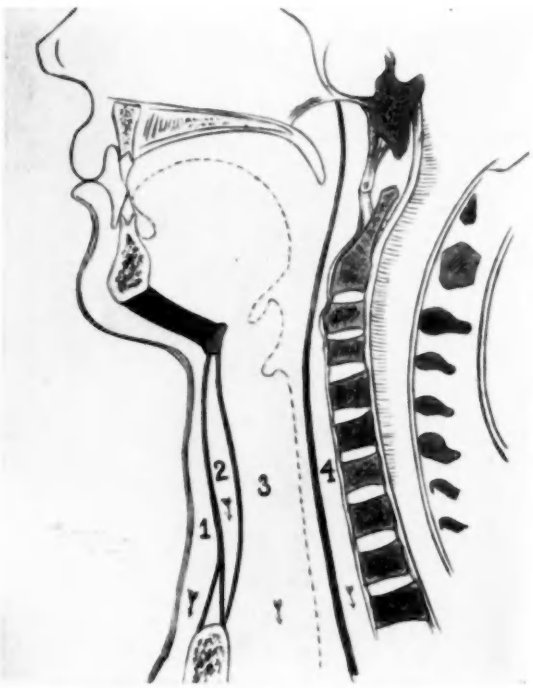


FIG. 6—To show the compartments made by the cervical fascia. Pus in Compartment No. 3 has access to the base of the skull, the pharyngo-maxillary fossa and the tonsil, the side of the tongue, the larynx, trachea and esophagus.

ting layer—so-called because it splits to enclose the trapezius muscles behind, the sterno cleido mastoid muscles on the side and the pre-tracheal or sub-hyoid muscles in front—is attached to the hyoid bone and takes a rest there, so to speak, before going on upward to complete its work. After getting a firm attachment

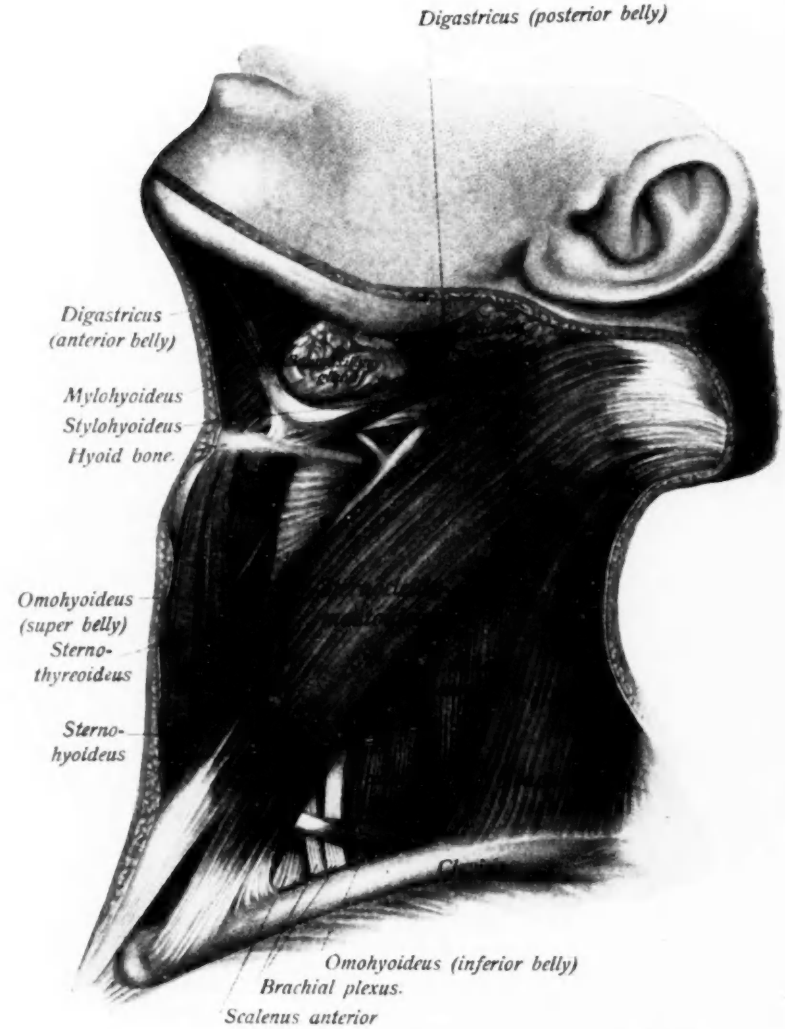
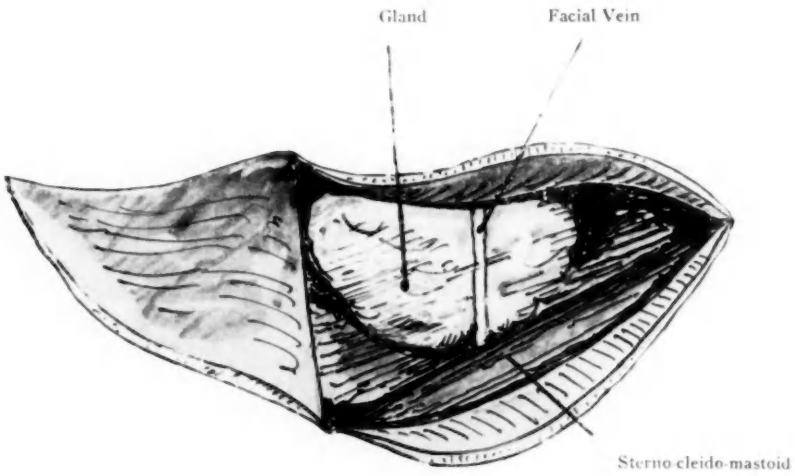


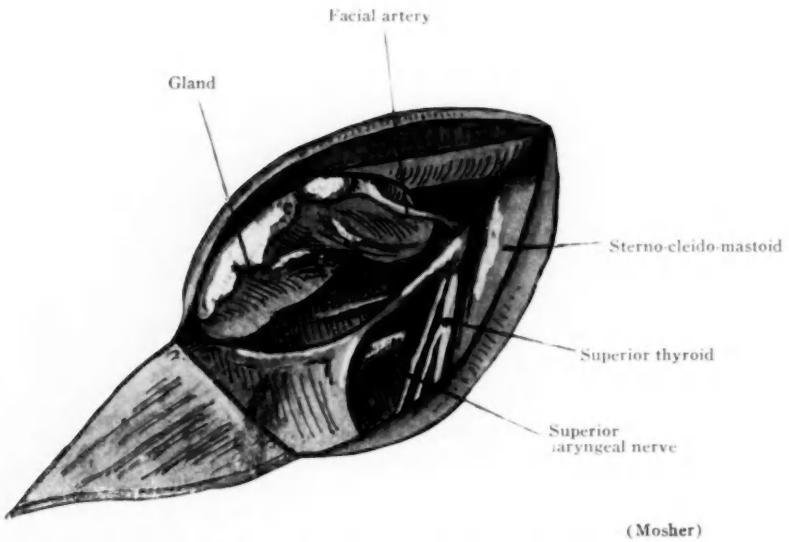
FIG. 7

(Sobotta)



(Mosher)

FIG. 8—Superficial dissection of submaxillary gland.



(Mosher)

FIG. 9—Deep dissection of submaxillary gland to show relationship about the tip of the great horn of the hyoid bone.

to the hyoid bone this fascia splits to enclose the submaxillary gland, the inner layer getting an attachment to the myloid ridge of the lower jaw and the outer layer to the outer edge of the horizontal ramus uniting at the same time with the fasciae of the parotid gland. The parotid gland has a fascial bed the same as the submaxillary gland and it is made in the same way by the splitting of this first layer of the deep fasciae. Superficially this layer is attached to the zygoma. The deep layer which goes under the gland gets an attachment to the styloid process and to a part of the under surface of the mastoid process. The deep layer of the parotid fascia thickens into a ligament which runs from the styloid process to the inner surface of the lower jaw as far down as the angle. This ligament separates the submaxillary gland from the parotid. The deep fascia of the parotid is not complete to the base of the skull. It fails to cover the upper inner surface of the parotid so that the parotid fossa communicates through this opening with the pharyngo maxillary fossa. The lower end of the parotid often extends downward below the angle of the jaw and encroaches on the submaxillary fossa. The lower end of the parotid gland with the facial nerve in it is easily turned upward. The upper part is firmly bound down.

The striking thing about the submaxillary fossa is its completeness. The gland lies in it like an egg in a nest. The facial artery enters the gland from behind but soon becomes superficial. When the gland is turned up the artery is carried with it.

The Pharyngo Maxillary Fossa

The pharyngo maxillary fossa or space, is more of a potential than an actual space until it is filled with pus. In the baby at birth it is easily demonstratable because it is filled with fat and stands out clearly. In the adult it may be considered as a cone, the base of which is attached to the base of the skull around the jugular foramen. The apex of the cone is at the hyoid bone. Reinforcing the fossa on the outside is the parotid fossa; below is the submaxillary fossa. The carotid sheath emerges from the apex of the cone and for practical purposes continues the fossa or cone through the neck into the thorax.

The inner boundary of the fossa is the superior constrictor with the tonsil attached to it. The outer boundary below is the internal pterygoid muscle lining the inner surface of the ascending ramus of the jaw and mating the masseter on the outside.

The outer boundary above is the parotid gland, the gland at this point not being covered with fasciae. Posteriorly the prevertebral muscles and the prevertebral fascia bound the fossa. The fossa is divided into unequal parts by the styloid process and the muscles

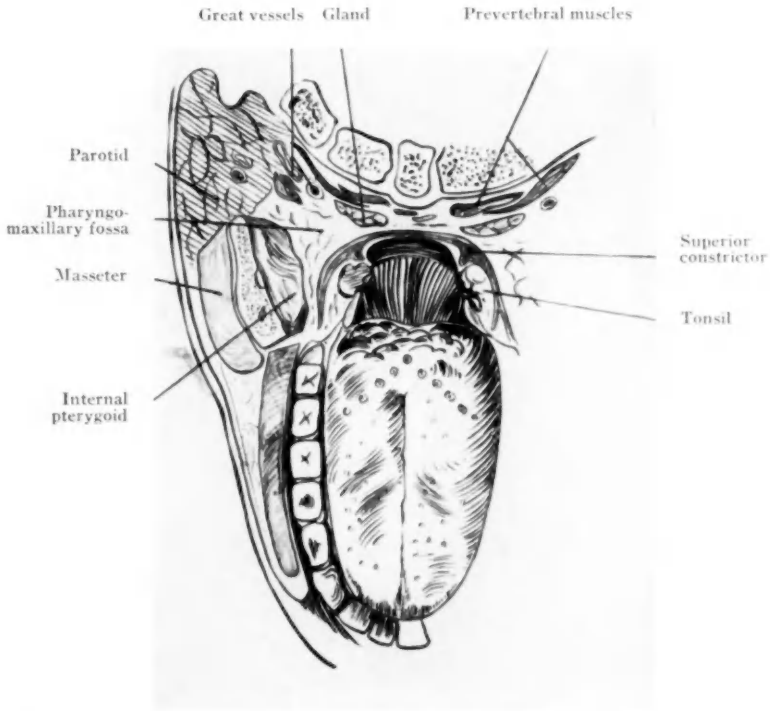


FIG. 10—Cross-section of the neck through the second cervical vertebra showing the pharyngo-maxillary fossa.

rising from it. These are in front of the great vessels and protect them. On the posterior wall near the middle line on the front face of the second cervical vertebrae there are two lymphatic glands which drain the nose and the upper pharynx and are in chain with the deep cervical glands.

The fossa from its anatomical position can be infected from the following structures: First and foremost the tonsils. Then from the glands in the fossa. These are the glands which suppurate in retropharyngeal abscess of infants. Infection can ex-

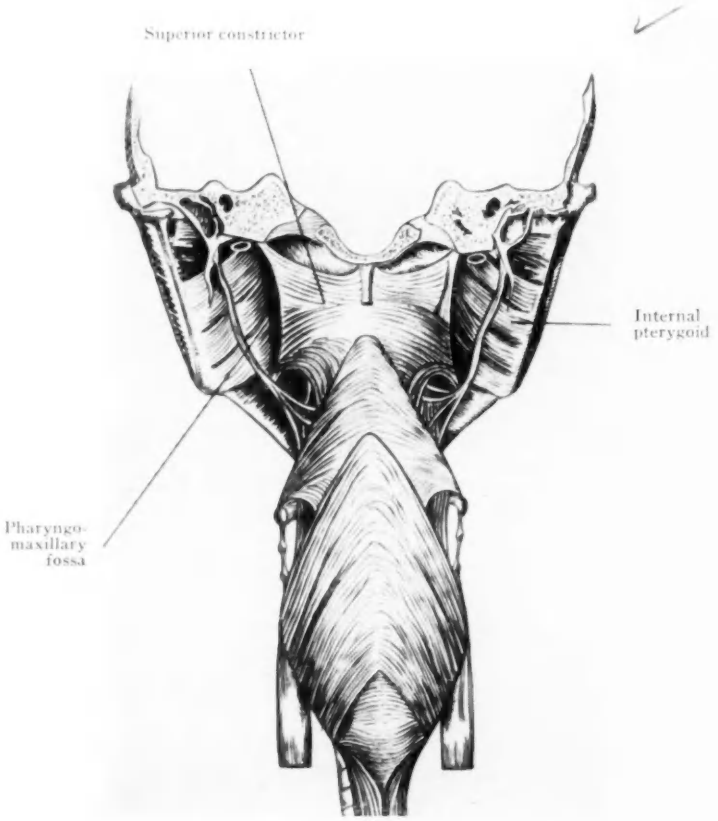
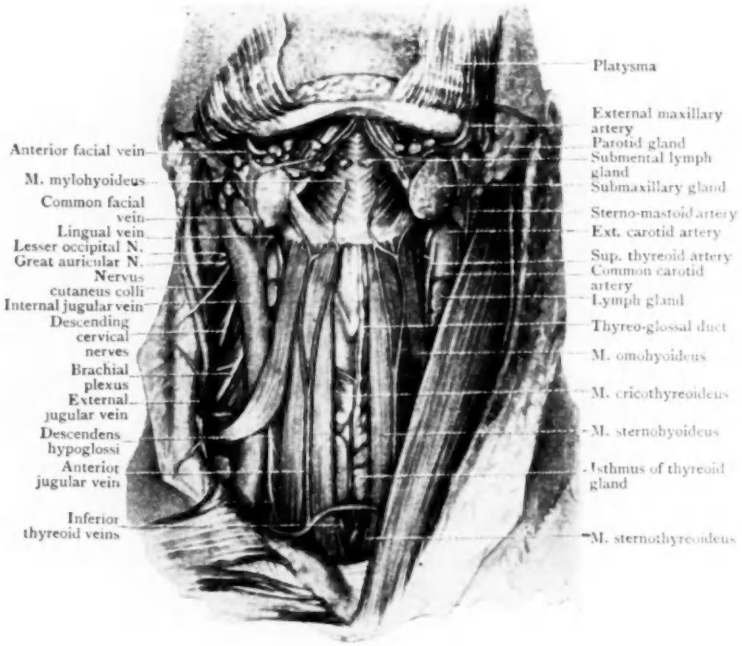


FIG. 11—Dissection to show pharyngo-maxillary fossa seen from behind.

H. P. Mosher

tend into the fossa from the parotid gland or from the tip of the mastoid—the so-called internal form of Betzold's abscess. Finally infection can come from the cervical vertebra. Such cases are usually tubercular.

The chief structure from the standpoint of infection in the fossa is the internal jugular vein. As I said earlier, unrecognized infection or thrombosis of the vein with general septicemia still takes its weekly toll of victims under the guise of blood-poisoning of unknown origin.



Dissection of the Front of the Neck. The Right Sterno-mastoid has been removed.

(Cunningham)

FIG. 12

For surgical purposes the side of the neck from the base of the skull to the hyoid bone is made up of the fossae just mentioned. One, the pharyngo maxillary, is a potential fossa until filled with pus; and the other two, the parotid, and especially the submaxillary, are actual fossae at all times.

The Veins

In order to work in a clean field in the neck the surgeon must see the veins first, not cut them first. They vary a little in position, but not enough to make this a good alibi. Everybody knows something about the internal jugular vein, and for some unknown reason also about the external jugular. Almost everyone, however, I find is hazy about the facial vein, the lingual and the superior thyroid veins. One seems to expect arteries, but not veins. The moral of this little preachment is to learn where to expect the veins and, as I just said, to see them first.

FIG. 13 (Toldt)

The facial vein and the lingual vein are especially important. Both are in relation with the tip of the great horn of the hyoid, the facial being below and the lingual above. In uncovering the internal jugular vein the superior thyroid veins give trouble if they are cut before seen, especially if cut short near the jugular. When this happens it is almost equal to cutting the internal jugular itself. The wound is flooded and everything becomes a mess. If the stump of the thyroid vein is cut too short you may have to tie the jugular before you can control the bleeding.

There is one vein which has confused me a number of times and I have seen it upset other operators. It is a branch of the anterior jugular which when present runs under the inner edge of the sterno cleido mastoid muscle. It leads to confusion when you are going to tie the internal jugular. You come upon it almost at once and think that you have the internal jugular. A glow of surgical satisfaction spreads over you and you look

FIG. 14

up to your assistants or to the visitors for the praise of the surprise in their eyes. Further dissection, however, shows that the vein, though reasonably large, is not large enough and is too superficial and not in relation with the carotid or the vagus nerve. Deeper dissection discloses the internal jugular with its classical relationships.

Tying the External Carotid

It has been proved that tying the common carotid does not have the great mortality due to interference with the intra cranial circulation which the older books state. Our recent hospital experience shows that it is reasonably safe.

Every now and then it is necessary to tie the external carotid artery. Any thing is, of course, justifiable in an emergency. It is, however, a bit mortifying always to have to find the common carotid and then work up to the bifurcation to find the external carotid. Not to be able to locate and tie the external carotid at least three times out of five without resorting to this round-about method is poor form. While speaking of the external carotid I wish to mention its first branch, the superior thyroid artery. This vessel is one which the surgeon should know how to tie or to avoid as the case may be.

This brings me to the tip of the great horn of the hyoid bone as the chief surgical landmark on the side of the neck. The books do not give it this importance. What I am giving you today is my own observation and experience, all respectfully and humbly submitted. I am always grateful for a landmark which can be both seen and felt. Such is the tip of the great horn of the hyoid. Out of sixteen major structures of the neck only three—all nerves—namely the glosso pharyngeal, the recurrent laryngeal and the phrenic, are not in relationship with it. For instance, the linqual vein is above, the facial below, the external carotid and the carotid sheath external, and if you follow the exposure which I like, the hypoglossal nerve is seen to lie in the field not above the tip of the horn but above the horn itself, and the superior thyroid artery and the superior laryngeal nerve are below. For good measure the little descendens noni is in the field on the carotid sheath. What more could you ask?

Incisions in the Neck

I have no patience with small incisions, especially in the neck. Cosmetic surgery and life-and-death surgery do not mix. The undertaker is the only follower of the medical profession who is justified in thinking of cosmetics first. In the neck the surgeon works at the apex of a cone. The deeper he gets the smaller his field. I am not advocating slashing for the brute pleasure of it. The scalp rather than the neck, is a safer place

for such exhibitions. I do like in the neck, however, a T-shaped incision when justifiable. For instance, in exposing the submaxillary gland I prefer a generous T-shaped incision which, when the flaps are held apart, gives a flat, open field. I like the same form of incision in exposing the larynx for thyrotomy or its complete removal. I use it also in a non-emergency tracheotomy under novocain. In this the flaps need not be large but

FIG. 15—To show T-shaped incision for exposing the submaxillary gland.

H.P.Mosher

their retraction gives a broad open field with the veins however placed standing out clearly.

Having unburdened myself a bit on incisions, I return to tying the external carotid. The incision is the typical one which begins just below the angle of the jaw and follows the edge of the sterno cleido mastoid obliquely down for about two inches.

If the neck is thick and deep I should turn down a rectangular flap. The object of the operator is to find the tip of the great horn of the hyoid. In getting over to this he avoids or ties the facial and lingual veins, as the case may be. To the outside of the tip of the great horn of the hyoid is the external carotid artery.

With what has just been said the first aim of this paper, namely the importance of the tip of the great horn of the hyoid bone, as a surgical landmark in the side of the neck, has been accomplished to the best of my ability.

The second and more important aim of the paper is to give you the increase of confidence in dealing with deep pus in the neck which I experienced when I realized the possibilities of the submaxillary fossa approach, in other words when I visualized the fact that the submaxillary fossa commands the side and the base of tongue, the floor of the mouth and the side of the neck from the base of the skull to the clavicle.

Before, however, taking up the submaxillary fossa approach I wish to say a word about abscess of the base of the tongue. There are two clinical types of abscess of the base of the tongue: the type which becomes superficial under the tongue and breaks and is comparatively innocent, and the burrowing or fatal type. Ludwig's angina I feel is often but an extension forward of pus from the base of the tongue. In one case I traced the pus by the submaxillary approach to a pocket opposite the tonsil.

To return to the submaxillary approach, a generous T-shaped incision will expose the submaxillary gland thoroughly and allow it to be turned upward out of its bed. This gives a wide open field which is most reassuring. The facial vein runs across the middle of the field and must be tied and cut. The submaxillary gland is turned upward out of its bed and the facial artery is turned up with the gland. The hypoglossal nerve is superficial and just above the V made by the tendons of the two bellies of the digastric muscle. The carotid sheath lies opposite the tip of the great horn of the hyoid bone. In searching for deep pus the submaxillary gland is kept up out of the way. The surgeon's finger then follows the carotid sheath upward or downward as his judgment dictates. As his finger travels upward it enters the pharyngo maxillary fossa and passes to the outside of the tonsil. Then the surgeon feels the end of the styloid process.

He carries his finger to the outside of this and reaches the base of the skull.

If the surgeon is dealing with pus in the floor of the mouth or in the base of the tongue an incision in the center of the floor of the sub-maxillary fossa can be carried forward or backward according to indications. The whole side of the tongue and the floor of the mouth are within easy reach. I cannot urge you too strongly to try this approach on the cadaver at your earliest opportunity. When I first realized its possibilities I had one of those surgical awakenings which come only too rarely but which stand out ever afterwards as milestones in your surgical career.

I have worked and taught with young men all my life and have often been asked about the ways and means of a medical career. I will put on record what I have to say on this topic as my closing paragraph. Last year Dr. Skillern called his chairman's address a sermon. In the hope that it may help some lonely soul before me I will speak in a similar vein and give what might be called my scientific creed. It is short and runs as follows:

If medicine is not your greatest fun, you are at the wrong job. Watch the clock and you will often miss the train. Overtime is opportunity. The cases from which you learn the most come on Sundays and holidays. The greatest satisfaction of a medical career is the acquaintance of the men who are doing things and being counted by them "one of the gang." Meet the busy men and take their overflow jobs—scientific jobs, I mean. Join a medical society of national scope and go to its meetings. Get a hospital connection. Never resign. Work to the age limit. It will come quickly enough. To spend a medical life doing nothing but private practice numbs the soul and kills the imagination. Set aside a stated time—I take an evening a week—for scientific medicine. Spend it in your library, the hospital library or in a laboratory. Come to an understanding with the family about it, peacefully if you can, by force if necessary. Read regularly something worth while besides medicine. Often, in such reading, words, phrases and even ideas which can be used in medicine will literally jump out at you. Make yourself better than the men of your community in at least one thing. What this medical thing is—notice I say *medical*—matters but little.

ADDRESS BY THE GUEST OF HONOR

SOME EXPERIENCES IN THE SURGICAL TREATMENT OF INFLAMMATION OF THE FRONTAL SINUS, AND ITS COMPLICATIONS

By HERBERT TILLEY, M. D., B. S. (Lond.) F. R. C. S. (Eng.)
Senior Surgeon, Royal Ear & Throat Hospital,

UNIVERSITY COLLEGE HOSPITAL, LONDON

Mr. President and Gentlemen: My first and grateful duty is to thank the Council of this distinguished Academy of Ophthalmology and Otolaryngology for asking me to be your guest of honour on this occasion.

You, Sir, were kind enough to suggest that I might relate some of my experiences in the Surgery of the Frontal Sinuses and of the more serious complications which may attend operative intervention for the relief of inflammatory conditions.

I accepted your choice because all pioneers are delighted to look back, and the inexorable finger of Time points to an article in The Lancet, September 26, 1896, entitled "An investigation of the Frontal Sinuses in 120 skulls with cases illustrating the methods of treatment of disease in this situation." Again, in The British Medical Journal, 1899, there may be found what I believe to be the first description of osteomyelitis of the frontal bone following upon an operation for chronic suppuration in its sinuses. Unfortunately, the subject of that communication died but I am able to show you the calvarium removed at the post mortem examination.

It would appear to be an almost unique specimen, but I trust that none of you will ever experience the misfortune of being able to secure a duplicate.

These early communications were published rather more than 33 years ago and by a lone young "tenderfoot" in otorhino-laryngology whose presence before you today he regards as a signal compliment to one of the early British workers in the revival of the surgery of the nasal accessory sinuses.

Before entering into any details of our subject it may be well to state some general principles or considerations which the surgeon must keep in mind when he undertakes the responsibility of treating acute or chronic inflammation of the frontal sinus by any form of surgical intervention.

First—He must have an accurate knowledge of the anatomy of the sinus, of its commoner variations and of its relation to important surrounding structures.

Second—He must never forget that inflammation is a protective reaction on the part of the tissues in their effort to resist and overcome a noxious invader, and therefore every detail of our treatment should aim at a minimum of trauma commensurate with such assistance as we may be able to afford Nature in her struggle for restitution.

Third—If this conception be granted, then it will be obvious that our primary object in surgical treatment will and must be the establishment of free, spontaneous, and permanent drainage of the products engendered by both invader and defender. I have said "free, spontaneous, permanent drainage"—four words—and would urge the younger members of our specialty to write them on the tablets of their hearts, and bind them on their foreheads.

In the treatment of inflammation of the frontal sinus our paramount difficulty is that we are dealing with an infected mucous membrane which lines an irregular and non-collapsible, bony-walled cavity from which drainage is impeded or, on occasion, completely obstructed.

To make this free and spontaneous has been the object of every operation yet devised whether it be by the external, or by the intranasal route. Had any one of them been uniformly applicable or successful, we should not be discussing the subject today. So we ask ourselves, "Wherein lies the difficulty with an external operation which is not so pre-eminent in any other inflamed bony cavity of the body?"

It seems to me that the answer is obvious, viz., the patient has a natural and instinctive dislike of a striking and disfiguring deformity.

Hence it comes about that our ideal operation must not only provide for free drainage—the basis of cure—but this must be attained with the best cosmetic result.

How can we secure these ends in acute and chronic inflammation of the frontal sinus?

Acute Inflammation

If we are fortunate enough to see the patient before the infection has destroyed the muco-periosteal barrier and entered the intimate bone structure of the sinus wall, it is often possible to encourage free drainage into the nose by the application of cocaine, adrenalin or ephedrine solutions to the lower end of the fronto-nasal duct where it opens into the anterior region of the middle meatus. Such a measure, possibly supplemented by scarification of the adjacent mucous membrane, will often suffice to give the desired exit for inflammatory secretions.

Should this fail, removal of the anterior half of the middle turbinal followed by frequent irrigation of the sinus with a warm hypertonic saline solution will often effect the freer drainage which is aimed at. If it be found that the naso-frontal canal will barely admit an ordinary probe, it may be well to slightly enlarge the canal and ostium in an anterior direction with a suitable rasp or burr. The value of irrigation is that it removes the irritating products of bacterial activity and of those incident to the inflammatory reaction of the mucosa. Such evacuation will often relieve the chief subjective symptom—headache—and what is also important, it will tend to reduce the oedematous obstruction around the fronto-nasal duct. When inflammation has passed into and through the bone to reach the periosteum it discloses itself by oedema of the soft tissues over the anterior wall of the sinus or of the upper eyelid, and in these circumstances it is unlikely that anything but external operation will suffice. This should not be unduly delayed because we can never say when a subperiosteal abscess may not bring about a septic thrombosis of an ophthalmic vein (superior or inferior) leading toward the cavernous sinus, with possible infection of the meinges.

External Operation

The type of incision for exposing the junction of the anterior wall and the upper and inner circle of the orbit is known to you all. When a disc of bone has been removed by a small trephine or by hammer and gouge, the aperature may be carefully enlarged by Citelli's or other suitable forceps, the limits of the sinus ascertained by a probe and its inflammatory secretions gently washed out by a cannula. If necessary, the ostium and fronto-nasal canal can be enlarged by a suitable probe or burr passed from above downwards. Three to four small drainage tubes should be

inserted so as to project through the middle of the external wound, and finally removed, when a mucoid discharge proves that the inflammatory process is practically at an end. For the first day or two hot fomentations should be frequently applied over the affected sinus because they relieve pain and assist in resolution of inflammation in the soft tissues. Under the influence of warm daily irrigations and later by injections of a 15 per cent argyrol solution the mucosa will gradually return to normal. The tubes can then be dispensed with and the external wound may be allowed to close.

It has been my good fortune not to have had a case of post-operative meningitis or of osteomyelitis follow the external operation for acute inflammation of the sinus, but I have published an instance of spontaneous osteomyelitis in an untreated case. (Brit. Med. Jour., July 7, 1917).

Certain difficulties may confront the surgeon in his endeavour to establish free internal (intranasal) and external drainage. I will mention two only:

1. A tortuous and narrow fronto-nasal canal. This one would attempt to overcome by a suitable burr passed from above downwards, or by removal of a portion of the ascending process of the maxillary bone and of the adjacent lacrymal bone.

2. A very large, irregular, and possibly loculated sinus. In such cases I should adopt a procedure which will be described later for the radical operation in chronic suppuration.

Warnings

(a) I can conceive of no circumstances in which a sharp curette should be used in removing acute or chronic inflamed mucous membrane from an infected frontal sinus. It is so easy to uncap the adjacent diplöe which are probably already inflamed, and by the admission of septic organisms risk a spreading and nearly always fatal osteomyelitis. If it be really necessary to remove masses of oedematous mucosa, this should be done with a mop of sterilized gauze or with blunt forceps such as those introduced by Luc (Paris).

(b) Whenever a general anaesthetic is administered for operation on the frontal sinus, the post-nasal space should be packed with a sterilized sponge in order to prevent blood and septic secretions passing into the lower air passages. Of course

intra-tracheal ether anaesthesia will obviate the need of such a precaution.

Chronic Frontal Sinus Suppuration

Before discussing the operative treatment of this condition, I would like to make a few statements which embody some personal experiences.

1. Headache is the most important and significant of the subjective symptoms. Its response to treatment will favour a good prognosis whereas its persistence after intranasal operation may raise the question of external intervention.

2. The removal of the ethmoidal infection which is practically always present, at any rate in the anterior cells, is the key to success in any type of internal or external operation. It is almost of equal importance to drain infected antral and sphenoidal sinuses before, or at the same time as the frontal sinus operation.

3. A practical point—it is often easier to pass a probe or cannula into a chronic suppurating frontal sinus than into one under normal conditions.

4. No operation should be undertaken without having studied frontal and lateral radiograms of all the sinuses. Their evidence may be the sole deciding factor in determining the particular type of operation to be employed.

Treatment

If an experience of more than a quarter of a century has taught me anything of this subject, it is that the number of cases of chronic suppuration of the frontal sinus which call for external radical operation are few.

In making this statement, let me say that I am not forgetful that a focus of sepsis in that situation has close anatomical relationships with the brain and its meninges and with the contents of the orbit. The purulent discharge may find its way into the larynx, trachea and lower air passages with the evil results we are familiar with—or it may be swallowed into the stomach with a sequence of gastro-intestinal disorders.

Carried by the blood stream or lymphatics the micro-organisms or their toxins may produce lesions in the large or small joints, in the cerebro-spinal nervous system, or failing such lodgment, symptoms of a general toxaemia may be revealed. A dark

enough picture indeed, if, in its contemplation we forget the existence in the body of Nature's immunising agents and her wonderful powers of defence.

In my earlier practice I saw many patients with chronic frontal sinus suppuration and often advised some external radical operation. Frequently such treatment was refused in favour of simpler palliative measures. Today, many of those people are old men and women who have enjoyed healthy, happy and useful lives. They visit me occasionally for inspection, for the solace of an optimistic report, or for some simple intranasal treatment.

It has interested me to note that in some of them, the discharge has entirely ceased, in others it is so slight as to form a thin, non-foetid crust in the anterior ethmoidal region which the patient can easily expel by a simple alkaline nasal wash. Had I performed a radical external operation on these cases, they would all have had some degree of scarring, probably the discharge would not have been cured in many of them, and in a few some serious complication might have supervened.

Such an experience seems to be in accord with that well known line of Tennyson: "Knowledge comes but Wisdom lingers," which is another way of stating that Youth fixes its gaze on possibilities, while maturer years constrain us to regard the broader field of probabilities.

We may now pass to a brief outline of the chief operations which have been introduced for the relief or cure of chronic suppuration.

For the reasons already given, it seems to me that unless there are definite indications to the contrary, the intranasal operation should be given precedence in our attempt to cure or to relieve the patients' symptoms.

In the average type of case these are headache, pain on pressure over the anterior or antero-inferior vault of the sinus, and not infrequently some degree of nasal obstruction caused by polypi or other high grade mucous membrane hypertrophies. The diagnosis is established by intranasal examination and supplemented by radiography.

The intranasal operation about to be outlined was introduced by Schäffer, developed by your countryman, Fletcher Ingals, and again by Halle (Berlin), and many others.

The Intranasal Operation

This involves removal of the anterior half of the middle turbinal at its junction with the "lateral mass," of any polypi or mucous membrane hypertrophies in the middle meatus and around the fronto-nasal canal, and possibly enlargement of the latter and of the ostium of the frontal sinus.

This seems a fitting opportunity for saying how much rhinologists must be indebted to you, Mr. President, for pointing out the surgical importance of the "agger" cells and for demonstrating how their removal may greatly facilitate the approach to the infundibulum and the establishment of freer drainage from the sinus.

For the enlargement of the fronto-nasal canal I employ a rasp with serrations on its anterior surface only, so that the canal and ostium are enlarged solely in a forward direction. Our chief difficulty lies in reducing the prominence of the frontal "boss" which forms the main hindrance to free drainage from the sinus. I have seen Halle (Berlin) use his electrically driven burr for this purpose, and was struck by the width of the communication which is secured. Having no personal experience of the method, it would be unfair to offer any adverse criticism of it, but fatalities have been recorded after its use and in any case I have an instinctive preference for an instrument manipulated by the hand and guided by that indefinable but intimate sense of touch. The last step of the operation will be the removal of any tags of mucous membrane or semi-detached spicules of bone. As free drainage is the great desideratum, no tampons of gauze should be inserted in the proximity of the operated region.

Three days later it is my practice to wash out the sinus with warm boracic lotion or a mixture of normal saline and peroxide of hydrogen. When the discharge lessens and becomes more mucoid, a 15 per cent solution of argyrol is injected after a preliminary irrigation with one of the above lotions.

For a few days after the operation the discharge may be profuse because the drainage has been improved, but if the headache has been relieved there should be no hurry to contemplate any external operation because it is reasonable to hope that the other symptoms will gradually abate, even though this may involve some weeks or months of waiting.

To the rhinologist who is skilled in intranasal surgery the operation is a comparatively easy one, and while serious and even

fatal complications are by no means unknown, they are much less frequent than with the more radical external operations. This fact, together with the absence of any scar or deformity, the short demands which the intranasal procedure makes upon the time of the patient, the great relief and occasional cure of the symptoms which may be expected from it—all these embolden me to make the statement that in seeking to "make the punishment fit the crime," with a few exceptions the intranasal route should always be chosen in the first instance.

In this profession of my faith I am glad to find that Prof. Hajek (Vienna) in the last edition of his classic work says: "I am convinced that in the majority of uncomplicated cases of chronic frontal sinus empyema, resection of the middle turbinal and the removal of hypertrophies is usually sufficient."

We may now ask ourselves, "What may be the conditions or the complications which would constrain us to advise a primary or a secondary external operation"?

PRIMARY EXTERNAL OPERATION:

1. A very narrow nasal cavity preventing free access to the ethmoidal region.

2. A tortuous fronto-nasal canal which proves to be impassable by a suitably curved probe, even after removal of the agger or other anterior ethmoidal cells in the neighborhood of the canal.

3. An extensive, irregular and loculated sinus. Not infrequently such secondary intrafrontal sinuses are associated with fronto-ethmoidal cells which spread outwards between the roof of the orbit and the floor of the frontal sinus. It will be obvious that the most perfectly executed endonasal operation could not provide effectual drainage for such conditions.

4. Attacks of subacute periostitis of the anterior or of the antero-inferior wall of the frontal sinus associated with tenderness on pressure over those regions and possibly with oedema of the upper eyelids. In this category one may place a chronic external fistula opening below the supraorbital ridge. Such conditions imply subperiosteal suppuration of the frontal or fronto-ethmoidal sinuses, and we can only deal with them effectually by an external operation which will enable us to remove carious bone and establish free intranasal drainage. On the other hand, I can recall a few cases of recurrent periostitis, one of chronic

external fistula in an old man, and two cases of recurrent iritis which were cured by endonasal operation, but I regard them as instances of good fortune and would only employ such a method in exceptional circumstances.

Time will not permit me to discuss the big and vexed question of the relationship between retrobulbar neuritis and frontal sinus or fronto-ethmoidal suppuration. I will only say that the nerve lesion is more likely to be associated with ethmoiditis than with the frontal sinus inflammation, and possibly the best results *qua* the optic nerve have resulted from operations on an apparently normal ethmoid or sphenoid sinus. Nevertheless, it would seem wise that, in the presence of a serious and progressive intraocular or retrobulbar lesion, an obvious focus of sepsis in any paranasal sinus should not be left untreated.

5. Symptoms of subdural abscess will demand a radical external operation and a suspicion of this complication may be strengthened by radiography of the frontal sinus regions.

Secondary External Operation:

1. When intranasal measures have failed to relieve the chief symptoms, e. g., headache and profuse discharge, especially if this be foetid.

2. The desire of the patient to be freed from the discharge which has not been cured or minimized by endonasal operation.

Other less common conditions may be symptoms suggestive of subdural abscess, or those which have been termed by William Hunter the "septic psychoses." You will probably add to this list the distal manifestations of sepsis as they affect the joints, the peripheral nervous system, the lower air passages, and the gastrointestinal tract, or the effect of blood-borne toxins on the general health.

External Radical Operations

What type of operation should be selected?

Let me say at once that there is no single operation which will meet the requirements of every case. It might be so if we could obliterate the sinus entirely as advised by Riedel, but cosmetic considerations will often preclude such a procedure. On the other hand, if radiography proves it to be of average size and shallow from before backwards, its obliteration will produce a cure with almost unnoticeable scar of depression. The opera-

tion entails removal of the anterior wall and the inflamed mucosa, establishing a free passage into the nose and encouraging the cavity to fill with healthy granulation tissue before the external incision is allowed to close.

Many of my most satisfactory results have been secured, in selected cases, by this method of obliterating a sinus. About six weeks of daily after treatment will be required for a sinus of average dimensions before complete healing has taken place by granulation tissue. This seems to me to be the point most open to adverse criticism. It resembles Coakley's operation, and differs from Kunt's procedure in that while I have only made one incision below the eyebrow and curving downwards towards the internal canthus, he cut a triangular flap in order to expose the anterior wall and inserted a drainage tube at the point where the vertical and horizontal incisions joined one another, and with the exception of this opening he sutured his incisions at the close of the operation.

I have had no experience with the Ogston-Luc operation, viz., opening the sinus at the junction of its lower anterior wall with the upper and medial wall of the orbit, curetting the mucous membrane and the anterior ethmoidal cells, placing a drain from the sinus into the nose, and suturing the incision at the close of the operation. The restricted opening does not give a sufficiently wide field for inspection of the sinus cavity, the establshment of free intranasal drainage must often be an almost blind manipulation and immediate closure of the external wound probably accounts for many of the fatal complications recorded in the literature of the subject.

Jansen preserved the anterior wall of the sinus but removed its floor before curetting away the mucous membrane. It is an excellent method for a comparatively small sinus and affords access to the ethmoidal cells, but in a large sinus with many recesses it is often difficult to reach these. Jansen acknowledges this himself and admits that it may be necessary to encroach on the anterior wall.

Riedel recommended the complete removal of the anterior wall and the floor of the sinus which is undoubtedly the best method for complete obliteration of the cavity and for exenteration of the ethmoid—but, as I have said already, the resulting deformity would frequently render such an operation impracticable.

Killian endeavoured to overcome these difficulties by his well known "bridge" operation. Many of us practised it, but in a sinus of considerable antero-posterior depth its obliteration by granulation tissue failed to take place, a dead space resulted and suppuration frequently recurred. Furthermore, not rarely the bridge necrosed and gave way with a resulting deformity which it was one of the chief objects of the operation to prevent. In favourable cases, however, the operation gave excellent results as regards the symptoms as well as a minimum of deformity.

Like most other operations fatalities occurred from meningitis or osteomyelitis. Hajek collected twenty-three recorded cases. Who knows how many more have remained unpublished?

In a large sinus with many recesses and extensive ethmoidal disease, the operation which is most frequently practised in London is that devised by Walter Howarth. (Journ. Laryngol. XXXVI. 1921. No. 9. p. 417). It provides for very free intranasal drainage by removal of the frontal "boss" and part of the ascending process of the superior maxilliary, easy access to the ethmoid and sphenoid sinuses and the cosmetic results are excellent.

To save time, I will show you the essential details of the operation by means of slides which have been taken from his own illustrations.

You will note that no attempt is made to remove all the inflamed mucosa because for its ultimate restitution dependence is placed on free, spontaneous, and permanent drainage.

My personal experience of the method is sufficiently satisfactory to prompt my recommending you to give it a fair trial, if you have not already done so. Having had no personal experience of Ritter's, Knapp's, and Lothrop's operations, nor of osteoplastic resections, it would seem a waste of time and scarcely fair to criticize them.

The Complications of Operating on the Frontal Sinus

It will be necessary to pass over all but a few of the less dangerous results in order to give more time to the serious risks of surgical intervention.

Diplopia is frequent after any operation which interferes with the pulley of the superior oblique muscle, but the trouble is generally temporary and in all my experience, in only one case has the symptom remained permanent.

Postoperative Orbital Cellulitis. This may complicate internal and external operations for acute or chronic suppuration, and is generally amenable to free incision and provision for drainage followed by hot fomentations until oedema tends to subside. In no instance have I known it to cause permanent damage to the sight.

Anaesthesia will of course always follow division of the supra-orbital nerve. Formication in the area of distribution of the nerve will last many months and some degree of anaesthesia will be permanent. I have never known division of the nerve produce more than a temporary neuralgia.

The two most serious complications because they are nearly always fatal are septic meningitis, and diffuse, spreading osteomyelitis of the frontal bone.

As a general rule meningitis follows from infection starting in the ethmoid bone, but as inflammation of that structure is to be the subject of a special discussion, we may leave its complications until then. It need only be said that acute meningitis may occur as a direct result of radical operations on the fronto-ethmoidal sinuses or it may prove to be the fatal element in cases of osteomyelitis of the frontal bone. My friend Logan Turner has lent me some slides which show how septic meningitis may arise by infective organisms entering the lymphatic sheaths of the olfactory nerve expansions.

Diffuse Osteomyelitis

This, the most serious complication of operations on the frontal sinus, has been so fully and ably discussed by my friend, Dan McKenzie, in the Journal of Laryngology, Rhinology and Otology for January, February, and March, 1913, that it would be impossible for me to improve on it even if time permitted my attempt to do so. It will suffice if I make a few general observations on the complication before discussing its prophylaxis and treatments.

Its chief characteristic is the tendency to spread beyond, and often far beyond, its source in the affected sinus. The calvarium I have shown you exemplifies this fact.

Staphylococcus (albus and aureus) or streptococci are the organisms most frequently found in pus from the inflammatory foci in the bone; the pneumococcus rarely, and in one case McKenzie established the presence of B. coli. He remarks that "so

far as the morphological type of the organism is concerned, no light whatever is thrown on the etiology of the disease." Whether such organisms gain access primarily into the osseous tissue around the sinus, or whether efferent veins are first infected and the bones around them secondarily implicated, are questions as yet unsettled.

From observation of the clinical features of osteomyelitis it is my impression that both portals share in providing access for infection.

But the riddle which we want to solve is this: How does it come about that experienced, skillful and careful surgeons may, after a long series of successful results, be suddenly brought face to face with this nightmare complication which may be followed by another run of happy results? For example, Killian performed his operation on 86 cases without accident, but lost three, one after the other, before his hundred were completed! It is difficult to believe that in those three fatal cases the infective organisms were peculiarly virulent, or that the resistance of the patients was out of all proportion so much lower than in the more fortunate cases.

Before passing to prophylaxis, I would like to state that I have lost one patient from a spreading osteomyelitis which started (after a tooth extraction) in the right antrum and spread upwards to the frontal sinus. After many extensive operations in the attempt to check its progress, the patient succumbed to a basal meningitis.

Another case followed a bilateral intranasal operation for chronic suppuration of the frontal sinuses. When these were opened up a chronic subdural abscess was found behind the posterior wall of the left sinus. This may have been the cause of what the patient called his "devastating headache"—the symptom for which the preliminary intranasal operation was performed.

Prophylaxis:

If we are to avoid this complication, I am convinced that:

(1) Before operation on the frontal sinus we should see that there is no untreated sepsis in any of the other sinuses, and especially of the antrum and the ethmoidal cells.

(2) Immediately before the anaesthetic is administered, the sinus should be thoroughly irrigated so as to minimize the risk of contaminating the exposed edges of the bony wound.

(3) When the sinus cavity has been exposed, any remaining purulent discharge should again be washed out.

(4) The less the mucous membrane is interfered with the better. High grade hypertrophies may be removed with blunt forceps such as Luc's, or possibly detached by small swabs of sterilized gauze. In no circumstances should a sharp curette be employed, especially in the upper regions of the sinus where already inflamed diplöe may easily be opened and infection admitted.

(5) At the close of the operation only the extremities of the skin wound should be sutured; the central portion should be left open for three or four days, and the loose stitches then tied.

Drainage can be secured by a parallel series of four or five small rubber tubes leading from the sinus to the middle of the skin incision.

Treatment:

When once recurrent suppuration takes place in the wound and an area of pale, putty-like oedema appears in the soft tissues overlying the neighborhood of (generally) the upper limits of the sinus, there must be no delay in making a free exposure of the frontal bone from the eminence to the affected sinus.

Free removal of the whole thickness must then be made with freshly sterilized instruments and commencing from above downwards, i. e., from healthy towards inflamed and infected bone. Only by such drastic measures have a few threatened lives been saved.

Let me warn the inexperienced that the earlier phases of osteomyelitis come almost as unheralded as a thief in the night. There may be little or no headache, possibly no pyrexia, or only a slight rise to 99°, and the patient may not experience any general malaise or marked local discomfort—all these come later and the surgeon must not feel secure in their earlier absence. The pale and painless spreading oedema around the wound must be to him the red flag of warning.

If and when such removal of bone has been made one or more isolated patches of oedema should appear beyond the field of operation, it will be obvious that the surgeon's effort has been forestalled by septic thrombosis of a diplöeic vein and further in-

tervention—though possibly worthy of consideration—is unlikely to be successful.

In these circumstances and dismayed with the sense of failure, he may be encouraged by the sentiments expressed by Tennyson in the last lines of his poem "Ulysses":

> "That which we are, we are,
> One equal temper of heroic hearts
> Made weak by time and fate, but strong in will
> To strive, to seek, to find, and not to yield."

SYMPOSIUM "BRAIN ABSCESS"

ABSCESS OF THE BRAIN FROM THE STANDPOINT OF THE OPHTHALMOLOGIST

WM. L. BENEDICT, M. D.,

Section on Ophthalmology, The Mayo Clinic,

ROCHESTER, MINN.

Rarely, if ever, does the ophthalmologist, in the capacity of a primary consultant, see a case of abscess of the brain. He rather is called on to interpret possible ocular phenomena in order to corroborate the diagnosis so far as these phenomena are determinate. The diagnosis of abscess of the brain is difficult even with ocular signs and symptoms, none of which is pathognomonic; all occur with other lesions of the brain, such as tumor of the brain, meningitis and encephalitis. There are also to be considered extradural abscess, osteomyelitis of the skull and tumor of the dura with softening or hemorrhage; these are conditions which may occur concomitant with diseases of the sinuses and the mastoid and give rise to ocular signs similar to those which are the result of abscess of the brain; they complicate the diagnosis. Although there is marked variability in the frequency with which ocular signs have been noted in cases of abscess of the brain, as reported in the literature, it is recognized quite generally that the absence of any ocular signs or symptoms makes the diagnosis less certain. It may be that the occurrence of ocular signs is reported as being more common by those who have access to ophthalmologic consultation, as some of the signs are elicited with difficulty or are evanescent in character and discovered only by repeated examination. In this country ophthalmology is combined with otology by a great majority of those engaging in the practice of these specialties. For the members of this Academy, therefore, it is impossible to view from distant standpoints a series of phenomena occurring in a case of suspected abscess of the

brain. It is possible to consider the significance of certain signs and symptoms of the visual organs and to correlate them with other data, but to base conclusions on ophthalmologic data alone would be sheer folly. Therefore ocular signs and symptoms must be considered in relation to all the data in a given case, the standpoint of the ophthalmologist being only a little aside from that of any other consultant on the case.

First to be considered, therefore, is the history of the onset and course of the pathologic process under consideration. Previous illnesses, accidents, operations, exposures, and habits, as well as occupation, habitual colds, headaches, and a host of other factors that might influence any symptom or sign or predispose the patient to intracranial disturbance must be carefully investigated and taken into consideration. The etiology of abscess of the brain is often obscure. As Kennedy stated, the diagnosis is fairly difficult in the absence of a direct lead from the etiology. The history of a discharging ear, previous mastoidectomy or a chronic disease of the paranasal sinus is often of primary significance in distinguishing abscess from other pathologic conditions and in determining the probable site of the lesion. It should also be borne in mind that multiple hematogenous abscesses may occur from a septic process elsewhere in the body, such as abscess of the lung or suppurative cystitis, while at the same time suppurative mastoiditis or disease of a sinus exists without direct relationship to the lesion of the brain.

One must also bear in mind the development of abscess of the brain, which was so well described by Macewen in 1893. He divided the clinical sequence of events produced by the formation of an abscess of the brain into (1) the initiatory stage, characterized by severe pain in the head, vomiting, rigor and fever; (2) the second stage characterized by pain in the head (which can be elicited only by percussion), slow cerebration, want of sustained attention, apyrexia, slow pulse, paralysis, and optic neuritis, and (3) the terminal stage in which there is either leakage of the pus into the membranes or ventricles, accompanied by fever, convulsions, and coma, or complete encapsulation of the abscess, which may remain quiescent for a long period and may finally become absorbed, in some cases without surgical interference, as illustrated in case 1.

Case I. A woman, aged fifty-two years, was referred to The Mayo Clinic in August, 1928, because of left exophthalmos

and diplopia. Her attention had been directed to a swelling and drooping of the left upper lid nine months previously and diplopia occurred two months later. During the seven months previous to her visit to the clinic the condition of the orbit had not changed noticeably. Four weeks before admission, a "diagnostic" opening had been made into the frontal sinus after the method of Haller. Mucus was not evacuated even though the roentgenogram showed marked pathologic change in the frontal region. Vision in the right eye was 6/5 and in the left eye 6/12; the fields and fundi were normal (fig. 1).

Fig. 1 (case 1). Mucocele of left frontal sinus; displacement of the globe downward and exophthalmos; empyema of right frontal sinus was present.

An incision was made through the brow on the nasal side down to the margin of the orbit and a quantity of thick, sebaceous material escaped. The fascia was separated from the orbital margin, allowing access into a fairly large frontal sinus which was completely filled with thick gray pastelike material. The wall of a cyst was found and removed. The wound was closed without drainage as the nasofrontal duct was large enough to admit a 7 mm. probe. There was no communication with the frontal sinus on the right side. The sinus was not deep and the roof was smooth and glistening. The floor was partly eroded, allowing for the pressure into the orbit causing the exophthalmos. The pathologist reported "hemorrhagic inflammatory tissue."

The patient was dismissed from the hospital on the fourth day. The following day the wound was opened, since the sinus evidently was not draining into the nose, and considerable pus came from the sinus. Drains were inserted into the wound

which continued to discharge pus. On the twelfth day after operation she complained of considerable headache. When she was lying on the left side or changing position from lying to sitting the pain about the left side of the head was very severe. She had a chill and the temperature rose to 100.8° F. She was again sent to the hospital and the wound was opened wide for better drainage. Considerable pus was obtained from two walled-off pockets on the temporal side of the wound. The temperature varied from 98.6° to 100° until on the sixteenth day it rose from 98° to 101°. The patient became stuporous and drowsy, refused food, and complained only of headache. She died of respiratory failure, the heart continuing to beat for a few minutes after respiration ceased.

Necropsy disclosed a rather large brain abscess of the left frontal lobe with perforation of the orbital plate and localized meningitis. Although the sinuses were not connected, the right frontal sinus was filled with pus. The abscess in the left frontal lobe was connected with the left frontal sinus, and by another small opening into the lateral ventricle.

The original pathologic condition was mucocele of the left frontal sinus, and purulent infection of the right frontal sinus. The brain abscess was not walled off but consisted of a pocket of pus on a line of infection extending from the sinus to the ventricles. This illustrated Macewen's third stage with leakage of pus into the ventricles although it was accompanied by slight fever, without convulsion or coma. Surgical interference would have been of no avail for there were no localizing signs and the abscess was not encapsulated. This type is sometimes referred to as fulminating abscess of the brain.

Clinically the ocular syndromes produced by encapsulated abscess of the brain are the same as those produced by tumor of the brain. When encapsulation has not taken place the process must be considered as one of inflammation or softening. The significance of this differentiation to the surgeon cannot be overemphasized. In The Mayo Clinic it has been noted that the best results are obtained by operation in cases in which the ophthalmologic and neurologic syndromes could not be distinguished from those produced by a tumor of the brain in the same situation. While the abscess is developing the choked disk fluctuates, becoming stationary when the abscess becomes encapsulated. In cases in which operation is delayed until encapsulation has oc-

curred, as shown by the disk, the postoperative course is less stormy and the mortality rate is lower. As a rule maximal choking occurs in a short time and surgical procedures may advisedly be deferred until the choked disc is stationary or has begun to recede. It is, therefore, in the second and third stages of abscess of the brain, as outlined by Macewen, that the ocular phenomena are of the most diagnostic and localizing value. In other words, from the standpoint of the ophthalmologist, the signs and symptoms of abscess of the brain are those of tumor of the brain. They are of great significance when present, but their absence is not conclusive.

The most common ocular sign of abscess of the brain is choked disk, present in about a third of the cases. A choked disk may not occur in cases of abscess of the brain or tumor of the brain; this depends on the size and especially the site of the lesion. The absence of a choked disk consequently is not proof of the absence of abscess; it is known that a certain type of abscess, the extradural, is not as a rule accompanied by choked disk. Lillie and Lillie reported from The Mayo Clinic four cases of choked disk associated with surgical mastoiditis; three of the patients had parasinus abscess and one patient had pachymeningitis with a large cholesteatoma pressing on the sinus and cerebellum. All the patients recovered and the choked disk receded. The same principle which, on the whole, is generally admitted for neoplasms has an effect in the abscess. The marked increase of the cerebral volume can be well tolerated without choked disk if the site of the lesion does not impede the intracerebral circulation of the spinal fluid.

Certain abscesses are favorably situated to produce choked disk, such as those in the temporal lobe; others, such as some cerebellar tumors, rarely cause it. Abscess of the frontal lobe, according to the consensus of opinion in the literature, causes choked disk infrequently unless the abscess is very large, yet in eleven cases of definitely encapsulated abscess of the frontal lobe, Lillie found bilateral choked disks ranging from 1 to 6 diopters, in eight. Abscess of the temporal lobe accompanied by choked disk is the most common, almost always arising from diseases of the ear. The degree of choked disk is also most frequently found to be greater, 4 to 6 diopters, in contrast to lesions of the cerebellum in which the swelling rarely exceeds 2 diopters; often slight blurring of the margins of the disk is all that is found. Choked

disk usually appears bilaterally and thus is of little localizing value. In many cases, however, the papilledema is not equal and cases are reported in which the lesion was found to be on the side on which the edema was greater. In other cases, however, the lesion was found on the side on which the edema was less. The reason for this probably lies in the occurrence of papilledema on one side earlier than on the other, or increasing edema of one disk occurring simultaneously with decreasing edema of the other. It is well known that choked disk does not occur in atrophic disks and that the papilledema recedes in an eye after atrophy is once well under way. In cases of certain lesions of the frontal lobe, direct pressure on the optic nerve may produce atrophy with or without previous choking of the disk, the basal frontal syndrome of Kennedy. Only one abscess so situated has been observed at The Mayo Clinic.

CASE II. A boy, aged fourteen years, was brought to The Mayo Clinic, October 27, 1926, because of headaches. Eight months previously he had had an ordinary cold in the head with fever. Eight days later the left eyelids began to swell and soon were swollen shut. Two weeks later vomiting began. The swelling of the lids disappeared and he returned to school after one month. In two days symptoms recurred, with swelling of the left eyelids and fever. He was relieved by the use of phenobarbital. Two weeks before examination the former symptoms recurred, with fever, headaches, and considerable drowsiness.

The general examination gave negative information. Roentgenograms disclosed cloudy frontal sinuses only. Vision of the right eye was 6/15, and of the left eye 6/60. The pupils and reflexes were normal. The fundi showed bilateral choked disk of from 3 to 4 diopters, with pallor of both disks and absorbing exudate. The fields showed concentric contraction for form and color. The blind spots were enlarged. There was a cecocentral scotoma in the left eye. The neurologic examination was negative (figs. 2 and 3).

Exploration revealed an abscess of the left frontal lobe. Subsequently, the vision improved to 6/10 in the right eye and to 6/15 in the left eye. The edema of the disks subsided to less than 1 diopter. The cecocentral scotoma receded to a small central scotoma in the left eye, and the blind spots were normal. Colors were recognized peripherally with both eyes.

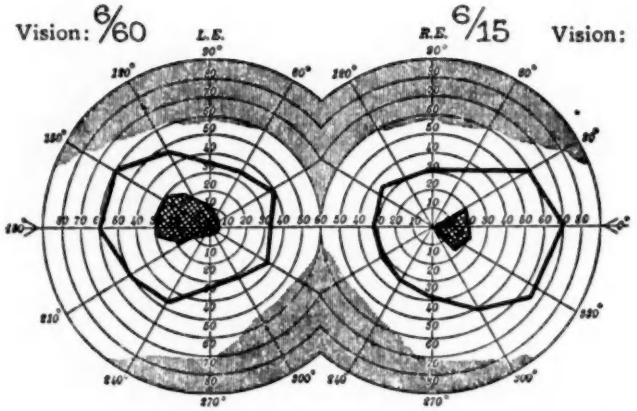


Fig. 2 (case 2). Fields on patient's admission: concentric contraction of fields; enlargement of blind spots with cecocentral scotoma of the left eye.

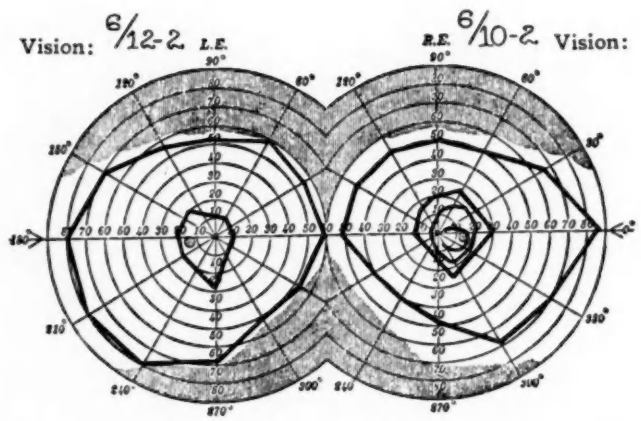


Fig. 3 (case 2). Two years after patient's admission; form fields showed slight contraction for both eyes; partial restoration of color fields; the cecocentral scotoma in the left eye had receded to a small central scotoma; blind spots were normal.

This is the only case observed in The Mayo Clinic of abscess of the frontal lobe with bilateral choked disk and a central scotoma on the side of the lesion.

Phenomena of peripheral vision have the same significance in abscess of the brain as they have in tumor of the brain. The most characteristic alteration is hemianopia and is dependent on the site of the lesion. Homonymous hemianopia appears in temporal abscesses if they are subcortical and affect the optic radiations. It is also found in abscess of the occipital lobe. Instead of complete hemianopia there may be only a quadrant hemianopia for colors, as occurred in case 3.

Case III. A girl, aged ten years, was brought to the clinic in May, 1928. She had had chronic otitis media in April. Since April 21 she had had headaches in the left temporal area. May 5, projectile vomiting with nausea developed. There was slight

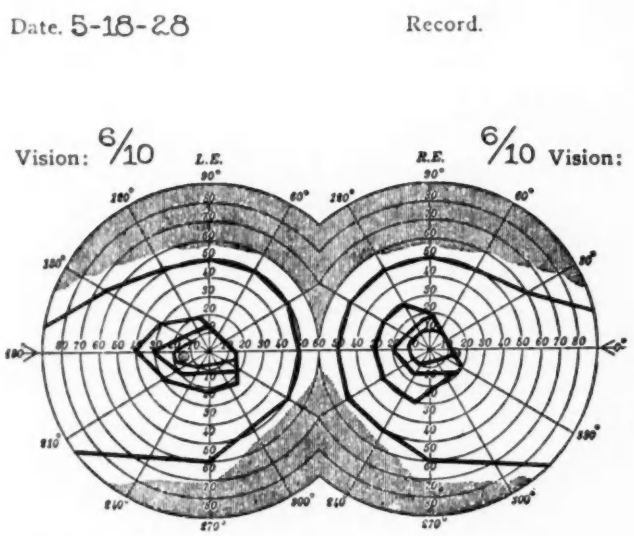


Fig. 4 (case 3). Slight contraction of form field with incomplete right homonymous hemianopsia for color.

bulging of the right eye and diplopia. Choped disk of 2 diopters in the right eye and of 3 diopters in the left eye were found. The vision was 6/10 in each eye. Fields showed right homonymous hemianopia for colors.

An abscess of the posterior part of the left temporal lobe was drained and the patient made a good recovery except for partial paralysis of the sixth and seventh nerves on the right side (fig. 4).

Characteristic field changes due to lesions of the frontal lobe are rare, and only occur in basally situated lesions. I have observed only one case (case 2) of abscess of the frontal lobe with bilateral choked disk and a central scotoma on the side of the lesion.

Complete or incomplete homonymous hemianopia with choked disk is common in cases of abscess of the temporal and occipital lobes, whereas in abscess of the cerebellum and abscess of the occipital lobe complete bilateral amaurosis has been reported, probably the result of secondary optic atrophy. Uhtoff stated that hemianopia accompanies 9 per cent of abscesses of the brain and that it occurs with greater frequency if the abscess is in the occipital lobe; such abscesses rarely originate in the ear. Hemianopia is sometimes the only symptom referable to the eye. Frequently, however, hemianopia is one of a number of symptoms among which choked disk is the most prominent.

Case IV. A man, aged forty-two years, had a history of having been injured while coupling cars. As a result of the injury, hemorrhagic suppurative cystitis developed. Subsequently he had a grand mal convulsion and on examination was found to have a few retinal hemorrhages in each eye. The vision was 6/6 in each eye; pupils, pupillary reflexes, and visual fields were normal. Six days later the vision was 6/6 in each eye, but there was slight horizontal nystagmus: both disks showed slight edema and there was left homonymous notching for colors in the upper quadrants.

A large mass, thought to be a tumor, was resected from the right temporal lobe. It proved to be a densely encapsulated abscess. The patient made a good recovery although hemianopia persisted (fig. 5).

Hemianopia is of no diagnostic value in the differential diagnosis of abscess of the brain, but is of great value in determining the site and possibly the extent of the lesion. In case of bilateral suppurative mastoiditis with symptoms of abscess of the brain, the visual field may be the only means of localizing the abscess. Nystagmus appears to be particularly common in cerebellar abscesses. It may be horizontal, rotatory, or vertical.

Disturbances of motility which are the result of paralysis of one or more of the ocular muscles is seldom seen. Abducens paralysis has been found both on the same side and on the contralateral side of the lesion in cases of abscess of the frontal lobe and lesions in the frontoparietal region.

Signs of partial paralysis of the third nerve are most often associated with abscess of the temporal lobe and nearly always associated with choked disk. Ptosis, paralysis of one or more of the extraocular muscles supplied by the third nerve, paralysis of the pupil, or lack of accommodation have been reported as oc-

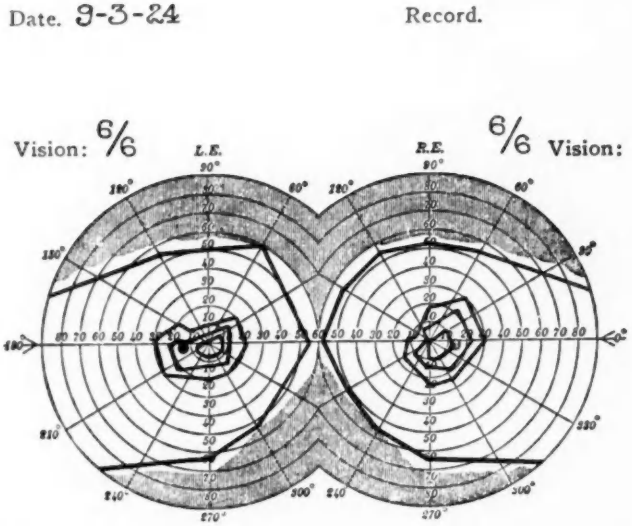


Fig. 5 (case 4). Bilateral left homonymous notching for colors in the upper quadrants; there was slight edema of the nerve heads; six days previously there was no edema of the nerve head and visual fields were normal.

curring singly or in combination. Paralysis of the third nerve is of considerable localizing value but paralysis of the sixth nerve may be concerned with only intracranial hypertension or inflammatory disturbances along the base of the brain, most frequently along the petrous portion of the temporal bone. It may lie far from the actual seat of the abscess.

Of the less frequently observed signs and symptoms, I have little to say because the observation is of little moment in the diag-

nosis or localization of abscess. Psychic blindness, visual hallucination, alexia, corneal anesthesia, and changes in visual acuity are not common.

Conjugate paralysis with or without tilting of the head may be present, but is of little localizing value. The syndrome of the superior colliculus of the corpora quadrigemia, fixed pupils, and paralysis of upward gaze indicate a lesion along the aqueduct associated with other signs of abscess, its occurrence is of considerable significance. It is frequent sequela of sleeping sickness.

Summary

The ocular manifestations that I have mentioned have been repeatedly noted in cases of abscess of the brain. Compared with data obtained at operation and at necropsy, their significance becomes apparent. Most consultants have learned to recognize many indescribable changes in a patient in whom characteristic septic fever, drowsiness, headaches, vomiting, psychic phenomena and visual or other ocular changes develop in the course of a suppurative disease, all of which point in a general way to involvement of the brain. The neurologist usually must decide whether there has been actual invasion of the brain.

The ophthalmologist, then, has two very important functions. The first is to aid in localizing the lesion; his signs are choked disk, changes in the perimetric field, ocular paralysis, and swelling of the lids and face. The significance of any of these observations varies with the dominant signs or symptoms in any given case. The second is to decide the proper time for surgical intervention. This is his most important service. An encapsulated abscess of the brain may be removed or drained with only slight risk whereas if operation is performed before the abscess becomes encapsulated the patient seldom recovers. Here the degree and course of choked disk is the most significant of the signs. When the degree of choked disk is stationary, the abscess, if present, is encapsulated, and the clinical picture becomes that of a tumor of the brain similarly situated. The ophthalmologist can then conscientiously urge surgical exploration.

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BRAIN ABSCESS FROM THE STANDPOINT OF THE OTOLARYNGOLOGIST

WELLS P. EAGLETON, M.D.

NEWARK, NEW JERSEY

Mr. President: I wish to thank you for the invitation to address this Society.

I have contended for a long time, in what teaching I do—for I am a clinician and not a teacher—that if I had anything to give, it was through some such method as the following: "Here is a patient with his history; we will now make an examination and from the history and examination, we will try to make a diagnosis and upon the diagnosis propose treatment."

But for myself, much that I have learned of real importance to me has come after I had made the diagnosis, and subjected the patient to treatment or operation, and the patient has died. I have then, at the post-mortem table, reread the history, reviewed the examination, and in the presence of the actual pathology asked myself the questions:

"Was my diagnosis accurate enough for surgical purposes?" "Was there anything in the history or examination that should have suggested a correct or more accurate diagnosis?" "Was the surgery adapted to the control of the condition?" "Could my surgery possibly have done him any good?" And on the other hand, "Is there any evidence that the trauma of the operation or the technique adopted was the deciding factor in his death?"

And further and most important: "What can I learn from this case to help me in the diagnosis and care of future patients?"

With these questions in mind I have brought to this meeting fourteen gross specimens of brains from patients who have died while in the Newark Eye & Ear Infirmary, or the Newark City Hospital; died as the result of suppurative intracranial complications of different kinds. With the specimens are the histories, which range from a complete history such as we should compile,

and do publish, down to the scattered memoranda of facts as they occur in our practice; the notes we make as we go along.

However, each specimen is accompanied by a complete report of all pathological findings observed during the post mortem. Each brain teaches a distinct surgical lesson.

With the aid of these specimens, I shall take this occasion to add a few important things that I have learned about brain abscess since 1922 when I published "Brain Abscess."[1] At that time I believed everything that I therein stated; but like everything else in life I now know that the book contains many errors.

Developmental Relationship Between the Intermedullary Blood Supply and the Meninges

A biological fact that has profoundly influenced my views in regard to all suppurative lesions within the dura was the demonstration that, during the evolution of life at the same period when a blood supply entered the nervous tissues,[2] [3] an intermedullary blood supply,—there developed a covering for the brain and for the nervous tissue itself. This relationship between the intermedullary blood supply and the brain coverings exist from the rays and sharks (Elasmobranchii) up to man. This intimate relationship contains the secret of many of the surgical and diagnostic problems that are under discussion. For examination of the human brain demonstrates that every cerebral vessel is surrounded by a prolongation of the pia-arachnoid—a perivascular space. Also all the veins within the dura have no outer coat, they are simply endothelial cells; the place of the outer coat, the adventitial coat of the extradural veins is formed by this perivascular space prolongation.

Further, this anatomical relationship is of great regional importance; for an examination of the veins within the dura—the cerebral veins—where they pass from the brain into the sinuses or extradural veins, shows that there are several points in the neighborhood of the ear and nose which, were they to become the seat of suppuration in the bone would bring infection in contact

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with the coatless endothelial veins, directly through the veins themselves or by their associations[4] with the venous sinuses.

"Points of Venous Vulnerability"

The minute veins that come out of the middle ear, and enter the sinus at the knee, and the veins of the vasa vasorum are especially important as routes of intradural infections. Two years ago I made a study of these veins and it considerably increased my mental horizon.

Venous channels and perivascular spaces are the chief routes of infection in brain abscess; for microscopical studies, show that the intima of the cerebral veins and the perivascular space surrounding the vessels are the two most frequent routes of infection into the cerebral substance. For brain abscess may originate from either a retrograde thrombophlebitis or a perivascular space infiltration.

A chill clinically differentiates between a blood stream invasion and a perivascular space invasion. Clinically the mode of invasion into the brain can be diagnosed and this may be of surgical importance for which infection from the ear travels inside of the coat of the vein, there is always a chill, if it extends by the perivascular space there is no chill.

I show the microscopical section from the brain (Fig. 1) of a man, with about the following history: He was operated upon in a general hospital for a right-sided mastoid suppuration. He then developed a lateral sinus thrombosis, which was also operated upon, following which he became slightly drowsy; he slept too much. He had a slight nystagmus on looking to the left but he had no papilloedema. His fields had been taken and they were said to be normal. Chart No. 1. But as he was dopey, he was transferred to the Newark Eye & Ear Infirmary, and the day he was transferred our technician demonstrated indentations of the visual fields. Chart No. 2. That same day he had a rise in temperature. Lumbar puncture showed the cerebral fluid to be infected. He died the day following the puncture. An autopsy revealed a hemorrhage, not only into the abscess, but into the lat-

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Reference: [4]Eagleton, W. P.—Cerebro-spinal fluid in the diagnosis and treatment of suppurative lesions. Opening Discussion British Medical Society, (Section Otolaryngology) Manchester, July 24, 1929, Jour. Otology & Laryngology, October & November, 1929.

Atkinson, E. M.—Abscess of the Brain: Its Pathology. Lancet 50, 483 (March 10) 1928.

eral ventricle and the pons, which gave him his nystagmus and caused his death. Fig. 1.

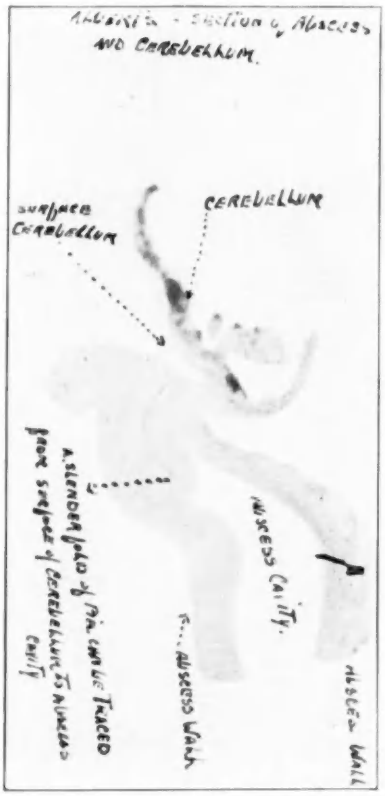

FIG. 1—Case of hemorrhage in choroid plexus and into lateral horn of ventricle, complicating temporo-sphenoidal lobe abscess. Post mortem report: "The dura is adherent to the posterior surface of the right temporal lobe. The adhering dura is continuous with the sinus when it makes the bend, and is directly over the area where the superior petrosal enters the lateral sinus.

There is a large abscess, the size of a lemon, which has ruptured in the posterior end of the right temporal-sphenoidal lobe. The contents of the abscess is hemorrhagic. There is a large vein running from the sinus to the abscess. There is a general encephalitis around the abscess. There is also a large hemorrhagic area in the posterior horn of the right lateral ventricle. (It was this hemorrhagic area that gave him Nystagmus). The hemorrhagic area is one inch long, and follows the large vessel of the choroid plexus, and nearly reaches the pons in the location of the basilar artery. Undoubtedly the perforation into the lateral ventricle caused death." Microscopical examination shows a retrograde thrombophlebitis through a small vein from the abscess. The meninges are densely infiltrated by leucocytes mostly polynuclear and contain large thrombosed vessels. A slender fold of pia can be traced from the surface of the cerebellum to the abscess cavity. The fold contains thrombosed blood vessels.

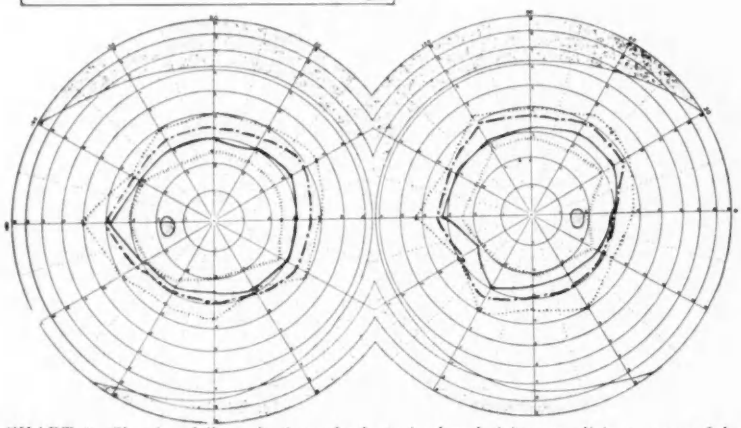

CHART 1—Showing failure, by imperfectly trained technician, to elicit a proper field in a drowsy patient.

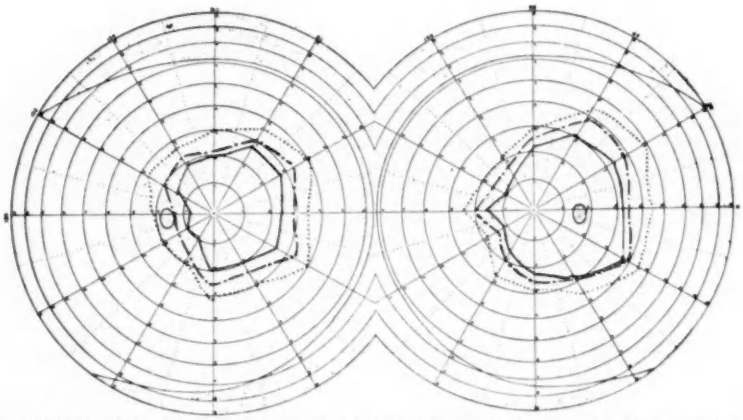

CHART 2—Fields of same patient taken by properly trained technician after repeated questioning. Patient still dopey.

Gross Hemorrhages in Brain Abscess

The inter-relationship between the perivascular spaces and the blood supply is still further shown by the frequency with which hemorrhages occur in suppurative diseases of the brain.

I show a specimen from a woman who had a cerebellar abscess. We explored and drained the abscess on two occasions. Suddenly while apparently doing fairly well, she became comatose and died in a few minutes from a large hemorrhage into the arachnoid at the base. Fig. 2.

I show a section from the brain of a woman on whom, in my effort to cure her of a profound sepsis from sinus thrombosis, associated with a meningitis the result of a labyrinthitis and a small brain abscess, I ligated the common carotid. Previously she had had intravenous injections of mercurochrome,[5] and as a result of the toxic action of the mecurochrome on the blood vessels and the disturbance of the circulation which the ligation occasioned, she had, on the following day, an interventricular hemorrhage. (Fig. 3).

And I have seen another woman on whom I operated and cured of brain abscess. Eighteen years later she went into coma and died; and as she became hemiplegic on the side previously paralyzed, I think that she had a hemorrhage into the old abscessed area.[6]

Reference: [5]Eagleton, W. P.—Cavernous Sinus Thrombophlebitis, 1924 Macmillan & Co., Case M, Page 163.

[6]Brain Abscess, loc cit, case XVII, Page 77.

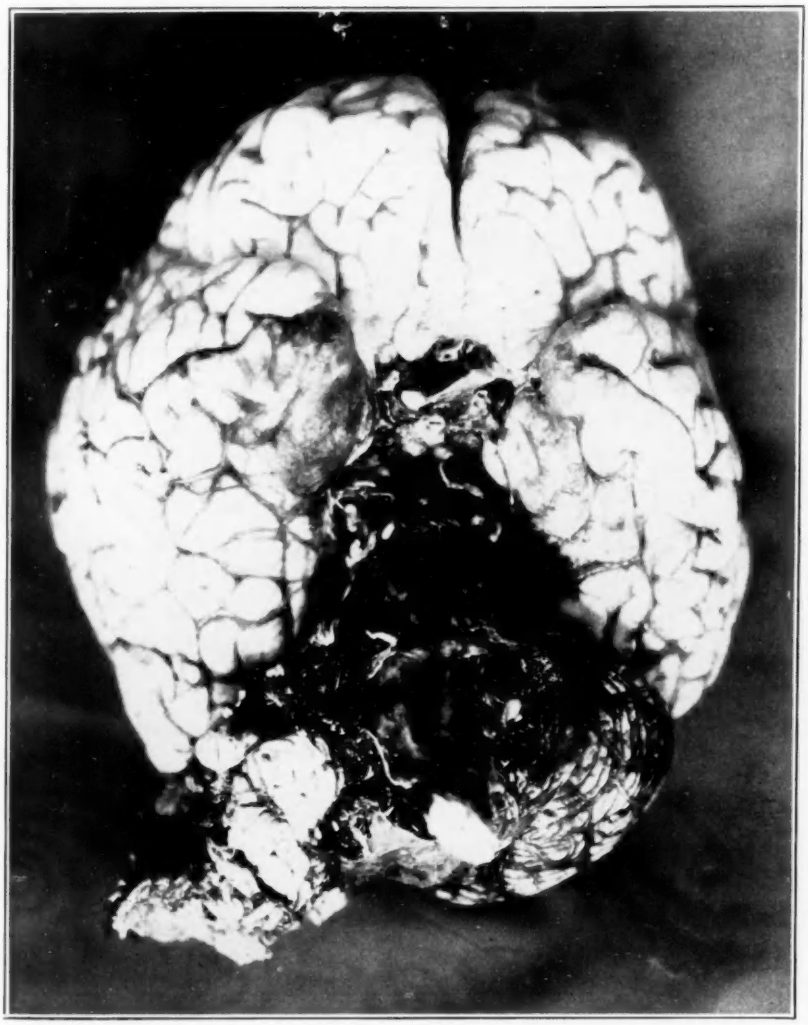

FIG. 2—Large subarachnoid hemorrhage filling basal cisterna and causing death; an unusual vascular complication of cerebellar abscess.

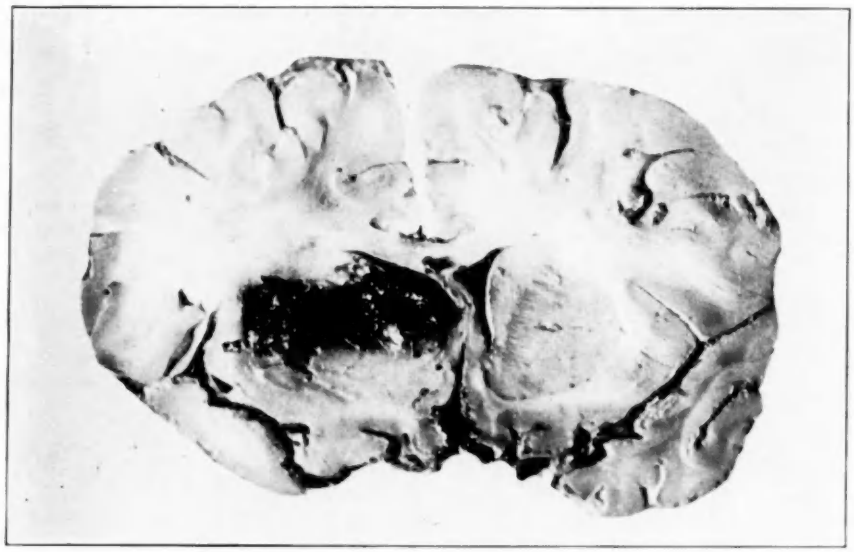

FIG. 3—Large intraventricular hemorrhage from a small abscess and ulceration of a branch of the middle cerebral. The intraventricular hemorrhage occurred 24 hours after the ligation of the homolateral common carotid; the vessel walls have probably been damaged by the intravenous injection of mercurochrome. The mercurochrome injection was followed by a chill, frequent urination and mercurochrome in the urine.

Specific Surgical Lesions in Intradural Suppuration.

What are some of the other lesions of vascular origin found in these fourteen specimens? First of all, the frequency of infarcts.

(a) *Infarcts.*

A child has earaches, it vomits and is ill. The doctor does a double myringotomy: one ear discharges freely; the other little or not at all. But the child continues sick; has temperature and vomits again and on the fifth day after the original earache, he suddenly goes into coma, becomes hemiplegic, has a lateral deviation of the eyes away from the side of the suppurating ear and has repeated convulsions. At autopsy we find a hemorrhagic infective infarct, undoubtedly from the ear, that had occluded one of the large cortical veins, around which occlusion there is an area of hemorrhage,—while the pia-arachnoid mesh of the cortex is covered by hemorrhagic areas. (Fig. 4).

FIG. 4—Hemorrhagic infarct in the midst of a general suppurative leptomengitis. The infarct is in reality a terminal manifestation of a virulent pneumococcic (Type III) meningitis. The meningitis had produced no alarming symptoms until the vascular occlusion and associated hemorrhage occurred.

Such infarcts are relatively common. They may get well—many do—if the vascular occluding nidus is not infected or when the infection is overcome by the protective reaction of the surrounding arachnoid.

To the latter class belong some of the so-called "pseudo-abscesses" in which the surgeon, in the presence of definite localizing symptoms, explores the brain and finds nothing. The operation, however, may be of some benefit, providing the operative

trauma is slight because it liberates the excess of cerebrospinal fluid which is secreted as the result of the "protective" reaction.

But if the surgeon is one of the brilliant hurry-up kind that poke holes in all directions through the dura in the brain in an effort to find a pocket of pus within the brain—the patient dies.

But septic infarcts do not always get well; nor do they immediately cause a severe meningitis as in the case of the boy.

An infarct in the substance of the brain may remain quiet for some time and then develop a brain abscess.

I show a section of the brain (Fig. 5) of an elderly woman who gave this history: She had had a left-sided chronic running ear for thirty years and recently had a convulsion. But her daughter said she had had convulsions a number of years before. A neurological examination revealed nothing.

Because she had repeated attacks of pain in the left ear in which the labyrinth had long ceased to function, we did a radical mastoid exenteration. On the fifth day after the operation, she had another convulsion, and on becoming conscious she showed a right hemiplegia and an aphasia. But the following day she was well; apparently all symptoms had disappeared. She went home — about three weeks afterwards — seemingly in good health. While at home her daughter said that she had some headache but it was not severe enough to cause her to return to see us. Suddenly she became hemiplegic and went into coma, she was brought back to the hospital;—an outstanding case of brain abscess. The abscess was evacuated but she died of hemorrhage into the surrounding tissue.

Infarcts, although far from uncommon, are but infrequently recognized. They may give all kinds of transient apoplectiform symptoms depending on the area disturbed by the vascular occlusion.

I saw a patient, in consultation with Dr. Walter Tymeson, who, several days after a simple mastoid operation followed by an up and down temperature, suddenly had repeated left-sided convulsions. On regaining consciousness there was demonstrable by the rough finger test a bilateral left hemianopsia which was complete up to the fixation point. However, on the following day the hemianopsia had disappeared from the field of the left eye leaving only an indentation in the field of the right eye—the side of the lesion,—this also soon disappeared and never recurred. **(Chart No. 3).**

FIG. 5—Abscess in neighborhood of internal capsule, the result of small septic infarct from a suppurating ear. The abscess did not develop for many weeks after the deposit of the infarct.

When however an infected infarct is deposited into the brain's substance it may be quiet and then later develop an abscess as in the case of the elderly woman (Figure No. 5).

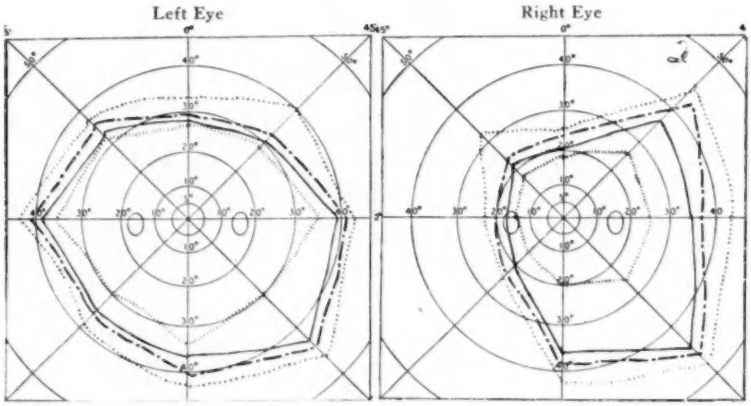

CHART 3—Left sided and upper indentation of visual field of right eye: the only remains of a complete bilateral left sided hemianopsia of the day before, in a patient suffering from a vascular occulsion (left sided convulsions) a complication of acute sepsis from mastoiditis and suppurative otitis.

(b) *Subdural Space Abscesses.*

The second surgically interesting pathological process revealed by these fourteen specimens is the frequency of abscesses localized in the subdural space. They have not been emphasized as they should be; for they are of frequent occurrence, especially over the frontal lobe. In my experience possibly 25% of all the frontal lobe abscesses are subdural space abscesses and if properly sought for, they should be easily located. A large osseous opening and a dural flap over the proper area, will disclose a flat collection of pus on the surface of the brain. It is apt to extend here, there and apparently everywhere. On the other hand the passage of a brain searcher through a small dural opening may fail to reveal pus. I have more than once, and that in recent years, overlooked a thin subdural collection—a so-called "superficial abscess," because I had explored through too small an opening in the bone and a nick in the dura.

In the temporo sphenoidal lobe subdural space abscesses are not common, however they are fairly frequent over the tentorium under the occipital lobe.

All cases of infection through the ductus endolymphaticus—so-called "empyema of the ductus endolymphaticus"—are in reality intradural space abscesses which are apt to become subdural space collections. Early recognized and properly operated on, ductus endolymphaticus cases should give a high proportion of recoveries.

I show a specimen from a case of sinus thrombosis* (Fig. No. 6) which had been operated on and was apparently cured, but later developed cerebellar symptoms. We opened an abscess in the subdural space of the posterior fossa, and accidentally perforated the dura of the middle fossa; pus escaped. The sinus infection, by direct continuity or a retrograde thrombophlebitis of a small vein, had originated a subdural space suppuration in both the posterior and middle fossae.

Cases of erosion of the superior semi-circular canal are apt to cause subdural abscess; and cases in which during a mastoid operation there has been an injury to the dura may develop subdural abscess. In the latter connection my experience shows that, if after drainage of a temporo-shenoidal lobe abscess the case does not do well, the possibility of a collection of pus in the subdural space incident to the operative opening of the subdural space is to be considered.

All cases of subdural space suppuration require large dural incisions as the infection may spread freely by continuity because of lack of adhesions between the inner surface of the dura and the outer layer of the arachnoid.

Aids to Diagnosis in Brain Abscess.

What have I learned about diagnosis?

I place more importance than ever, in diagnosis, upon the probability that certain areas in the primary focus of infection if diseased—that is caries in Trautman's triangle; labyrinthitis; perisinus obscess; granulations *over* the sinus low down towards the bulb; jugular bulb thrombosis—are apt to follow definite paths when the suppuration extends. That is the importance (1) of *anatomically known routes through the dura* by pathological and anatomical preformed ways in the arachnoid and into the brain:

Consequently, the importance of (2) the *site of the original infection** as found during a simple or radical mastoid operation performed at a time when there are no brain symptoms; because if brain symptoms occur later, the pathological change revealed by the primary operation may suggest what to do and where to go.

Reference: *Eagleton, W. P.—Localizing Value of Ophthalmic Examination in Suppurative Diseases of the Brain—Jour. A. M. A. Mar. 2, 1929, Vol. 92 case 6.

*Eagleton, W. P.—Diagnosis of Intracranial Complications, Page 578 in Jackson & Coats: Nose, Throat & Ear and Their Diseases, Saunders 1929.

FIG. 6—Subdural space suppuration in the posterior fossa with an associated middle fossa subdural space collection of pus both originating from a retrograde thrombophlebitis of veins from an infected lateral sinus. Mistaken for an intracerebellar abscess.

A Non-Functioning Labyrinth in Diagnosis.

The labyrinth stands in the middle of the petrous pyramid, so all osseous paths of infection are either (a) extra labyrinthine, (b) intra labyrinthine or (c) in the petrous apex. Consequently, of prime importance in the localization of the site of an intracranial lesion is to determine, "Is the labyrinth functioning?"; and if not, "when did it lose its function?" For if the labyrinth has recently ceased to function the path of infection is probably through the labyrinth and has entered the dura by the internal auditory meatus.

(3) *The Importance of Vomiting?* I know that some of my friends think I am extreme in this, but I believe that given a suppurating ear, if the patient vomits, something has happened within the dura. Of course, it is possible to have an abdominal cause, but ordinarily, especially in children, unless there is a manifest reason, vomiting during an otitis media, should arouse the suspicion of an intracranial complication.

Again our experience teaches that in a considerable number of cases the times of invasion dates from an initial vomiting. The case I showed, (Fig. 4 the child with pneumococci infarct) had an initial vomiting which was one of the manifestations of a localized meningitis and which was probably curable at this time by the complete exenturation of the primary focus in the mastoid, or even during the next day or so if the mastoid surgery had been associated with evacuation of an adjacent collection of infected cerebrospinal fluid. Five days later, however, when there were septic infarcts and general suppurative leptomeningitis, the case was hopeless.

(4) *Routine Visual Fields.* We have a visual field of each eye taken every day. It is a tiresome procedure but I have learned that the chief manifestation of *the oedema which accompanies a brain abscess may be alterations in the visual fields.*† However, like all oedemas, the indentations may vary from day to day; they may be present one day but gone the next day. The only time when fields can be properly taken is when there is only a possibility that an abscess may be present; when the intracranial lesion is manifest the patient is usually so drowsy that he cannot cooperate.

I exhibit seventy-eight fields taken on one case of brain abscess that recovered.[2] The case is remarkable in that this boy's brain was explored and drainage instituted on four occasions; each time largely because the sudden alterations in the fields suggested that the suppurative process was again advancing. The patient himself came to appreciate when he was not doing well by the condition of his fields, even in the absence of other symptoms; he would say, "Doctor, my fields are in today. I suppose you will be operating on me again."

But homonymous indentations of the visual fields may not be present and still there may be extended oedema of the brain, provided there is no intradural pressure. This may occur when a decompression operation has been performed. For it is the pressure upon the visual tract by the oedema of cerebral tissue confined within the dura and skull that causes the indentations.

Reference: †Eagleton, W. P.—Localizing Value of Ophthalmic Examinations in Suppurative Diseases of the Brain. Jour. A. M. A. Vol. No. 92, page 713.

[2]Loc cit case 4.

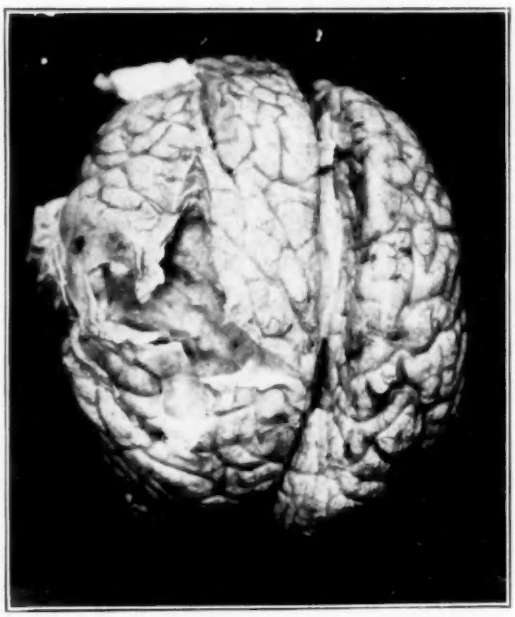

FIG. 7—Large abscess in left frontal, parietal and temporal lobes but causing no hemianoptic indentation of fields because of extensive decompression of left frontal region.

I show the brain of a man (Fig. 7) who had a left frontal sinus suppuration, then a cavernous sinus thrombophlebitis and a meningitis. I ligated the common carotid, eviscerated the orbit and drained the septic cavernous sinus to control his blood stream infection; I then removed the bone over the whole left frontal area and made a small opening in the dura; thus giving him a large bony decompression and evacuating a collection of septic fluid to combat the localized meningitis. For a long time he did well. But he had a slight aphasia at times. He also was depressed; but he had no headache. We took many visual fields, and largely because they all failed to show any indentations he was allowed to leave the hospital after several months. (Fig. 8). Three months later he returned with headache, vomiting and papilloedema. We explored and found an arachnoid cyst; and we again thought we had helped him. However, one day he went into deep coma, and he died of a very large abscess which had failed to cause indentations of the visual fields because of the extensive decompression.

FIG. 8—Large frontal bone defect lower one-half of which was granulating mass several months after operation, following evisceration of orbit, draining of cavernous sinus, and evacuation of septic fluid from meninges. As the result of the extensive decompression there were no indentations of the visual fields although a large abscess was present. See Fig. 7.

(5) *The Value of Nystagmus.* I think those of us who do this work, should throw out of our minds much that has been taught about the localizing value of the direction of the nystagmus. I am speaking now of intracranial lesions, not labyrinth disease. We should ask ourselves: Has this man a spontaneous nystagmus, or has he not? We know too little about the central connections of the vestibular apparatus to befog the issue by clinging to theories that we now know are wrong.[1] I am sure that we frequently confuse and deceive ourselves in trying to make a diag-

Reference: [1]Vestibular Tests in Cranial Surgery, 2nd paper. Read before American Neurological Ass'n, Washington, D. C., May 6, 1925 (Unpublished).

nosis of the *location of the lesion by the direction of the nystagmus.*

There are a few points, however, that have stood me in good stead about nystagmus in suppurative lesions.

(a) Vertical nystagmus either spontaneous or induced suggests a brain stem involvement.

(b) A changing spontaneous nystagmus suggests a retrolabyrinthine origin.

(c) A vertical diplopia associated with or *followed by a spontaneous nystagmus*[2] suggests a very deep, a mid-brain collection in the anterior midbrain segment of the posterior fossa—mesencephalic.

Of abnormally induced nystagmus, the most important in acute suppurative lesions is that during a cold caloric is (d) great diminution or absence of nystagmus in the upright position especially of the contralateral side associated with a normal nystagmus in the horizontal position* is diagnostic of increased intracranial pressure.

But they are all only suggestions; they are not really localizing, for the nystagmus may originate from a lesion above the tentorium.

I exhibit the brain of a man who was referred to me by Dr. Moorehead of Brooklyn. This doctor had done a very clever thing. He said this man has brain abscess, and when I asked why, he said: "Because his tongue has remained coated too long, and now he has a little nystagmus and has a pain in the head." I thought "that is easy—he must have a cerebellar abscess;" but when I examined the patient, it was not so easy. He had had a double mastoid—the right lateral sinus had become in-

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[2]Localizing Value, etc. loc cit, case 7.

*Eagleton, Wells P.—Vestibular Tests in Intracranial Surgery. (First Paper). The importance (1) of the absence of reactibility of the vertical canals of both ears to stimulation by the cold caloric in the diagnosis of increased intracranial pressure of the posterior fossa, and (2) of the reduction of the duration of the nystagmus from turning in diffuse lesions of the cerebrospinal system pathways over the cerebellar cortex (as in protective meningitis and linear fractures of the skull of the posterior fossa). Published in American Laryngological, Rhinological and Otological Society, Inc. 1922.

fected and had been operated upon. But he now had pain in the left occipital region and a changing nystagmus. We decided that he had a cerebellar abscess probably on the right side although his pain was over the left occiput and in the left eye which made a left-sided lesion a possibility. We did a double cerebellar decompression and explored the cerebellum and found nothing. The first day after the operation, the nystagmus disappeared. But the man did not improve. His fields were not indented; but suddenly (when he was almost unconscious and somewhat irrational) the technician said she though he *could not see on his right side.* That suggested that the abscess was not in the posterior fossa but in the middle fossa and *on the left side.* He died, and the specimen shows the abscess in the occipital lobe of the left side.

Mistakes

Now what have we learned about what we should not do? *Danger of lumbar puncture and ether in brain abscess.*

I do not do a lumbar puncture in any case in which there is even a suspicion of an abscess which may involve either the posterior fossa or any part of the midbrain cone that passes up through the tentorial ring. I exhibit the brain of a man who I thoought had a meningitis and on whom we performed several lumbar punctures and two ventricular taps. Sudddenly after a lumbar puncture he stopped breathing. At post mortem we found a small hemorrhage into the arachnoid over the medulla that we had caused by the last lumbar puncture.

Last of all, we do not give our cases ether. We try to do all operative work under local anesthesia. I show the brain of a man sent to me by Dr. Lynn Emerson of Orange. His history was that he had had two running ears for some months, then they ceased to discharge and now he had pain in his head, vomiting, nystagmus and papilloedema. His fields were not indented. We tapped both ventricles, and while I was "playing with him" he became so irrational, that he could not be controlled and he was given ether preparatory to a cerebellar exploration. Suddenly he stopped breathing, and although artificial respiration was performed for five hours and although with a direct laryngoscope his cord were seen to move with each artificial filling of the chest, he never breathed again. At autopsy we found a small abscess (Fig 10) in the arachnoid mesh near the angle; and later another

small abscess near the mid line which was so hidden that we did not find it until we had hardened the brain—a little abscess in the substance of the brain stem, that had given tremendous symptoms and caused death by respiratory paralysis when ether was administered. (Fig. 9).

Operative results:

What has been our mortality during the last seven years in the treatment of brain abscess? I must confess that in temporo-

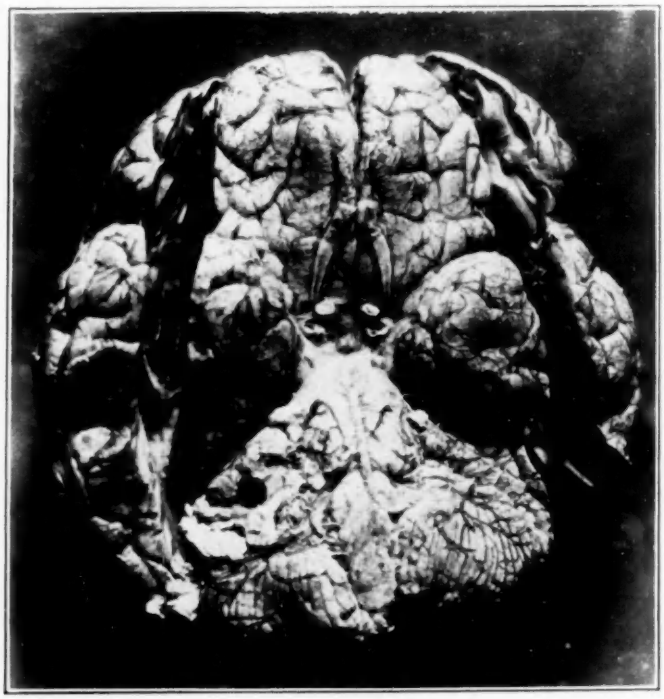

FIG. 9—Area of encapsulated pus in the meshes of the arachnoid marked by black disc.

sphenoidal abscess our results have not been as brilliant as those reported in the literature, nor have they greatly improved over those of former years. This I think may be partially due to my having abandoned the large bony flap that I orginally employed.

But in the very nature of the pathological process and its anatomical situation,—an abscess within the temporal lobe must be a "killing" disease in a large proportion of cases. For it is a destructive suppurative space consuming lesion, confined within a

FIG. 10.—Same brain as Fig. 9. *Encapsulated abscess possibly tuberculous, in brain stem* so small that was not discovered at time of post mortem—respiratory failure on administration of ether.

bony cavity; and situated not only in the highest organized tissue of the body—the brain—but in the latest addition to the neopallium—the spheno-temporal lobe—in the neighborhood of the basal ganglia and the central ventricular system none of which can be displaced without serious damage to the ganglia or extension of the suppuration into the ventricle. Consequently although a temporo-sphenoidal lobe abscess is in a symptomatic "silent area," the above unfavorable factors make such an abscess especially liable to a surrounding encephalitis and infective thrombophlebitis;

while its proximity to the ear, renders a relighting of infection in the damaged cerebral tissue, a constant possibility.

I wish we followed all our cases of so-called cures of abscess of the brain for three years at least; the same as they do in cancer cases. I think we would be surprised to find that several had died with cerebral symptoms. Such has happened to two of my cases*; in which, after over a year of apparent health, operation or post mortem* revealed an area of suppuration within the brain which had been quiescent until reinfected.

But our results in cerebellar abscess have markedly improved, — over 50 percent have recovered. This is largely due, we think, to the extent of the initial bony decompression; for the first thing we do is to remove all the bone covering both cerebellar hemispheres from the opposite lateral sinus to the diseased petrous pyramid and from the transverse sinus above to the foramen magnum below;[1] and as this is followed by "unlocking" of the petrous pyramid[2] with removal of the angle of the superior petrosal and of a large part of the squama, the bony decompression is very large.

Also intradural suppuration of the frontal region has given much better results than before. Histologically this should be so, for here we have a localized suppuration in a "silent" area from the ventricular system and basal ganglia, without firm connections to the dura and thus amenable to considerable displacement without damage to the surrounding parts. In addition to this the proper short circuiting of the mucous membrane† of the

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Reference: *Eagleton, W. P.—The Operative Treatment of Suppurative Meningitis with especial reference to irrigation of the cranial and spinal subarachnoid spaces and the importance of protective meningitis from a prognostic and therapeutic standpoint with an analysis of the cases of recovery—exclusive of meningococcic—reported in the literature. From American Otological Society, 1921 (Presidential address) Case VII, Page 52. "Cerebral and cerebro-spinal subarachnoid irrigation for general suppurative meningitis (streptococcic, hemolytic) originating from a latent and unsuspected cerebral abscess from a gunshot wound traversing the frontal lobe eighteen months previously."

[1]Eagleton, W. P.—Operative Treatment of Cerebellar Abscess, Surgery, Gynecology and Obstetrics, November 1924, page 653.

[2]Eagleton, W. P.—Cerebrospinal fluid as an aid to diagnosis, etc., Jour. Laryngology and Otology, November, 1929, Page 734.

nose should render a later infection much less liable than in the temporal lobe.

Lastly, our results in meningitis have been such that we have been more than encouraged. In the last 107 cases that Dr. John V. Bissett and myself have operated upon, 62 have recovered—about 60%.

Today if we have a case of abscess of the brain which develops meningitis, we pay little attention to the abscess, but try to cure the meningitis. For, as long ago we learned that when a patient with a chronic running ear develops intracranial symptoms, not to perform a radical operation, but to attack the intracranial lesion; so recently we have realized that in meningitis resulting from an abscess the meningitis should be first controlled, and this, if successful will allow of the drainage of the abscess with less risk to the patient.

Conclusions:

In conclusion, I want to emphasize: (a) every otologist should, for his own sake, make a careful note of each pathological finding in all cases of mastoid; and later if intracranial symptoms develop try to think anatomically. With infection in such and such a place, he should try to visualize what has happened inside the cranium; what course the process should take in its passage through the dura, and within the arachnoid or cerebral tissue. (b) Vomiting in any case of a running ear should make the surgeon suspicious of an intracranial complication. (c) Every chill that occurs in an intracranial complication is a sign of blood stream infection although the infection may be in one of the veins of the cerebral tissue not necessarily in a venous sinus. (d) Have somebody take visual fields daily. It is a difficult task, do not try to do it yourself or it will not be done. Do not ask an ophthalmologist to do it, for he will do it but once. But have someone whose calling it is to take fields routinely, carefully, and intelligently; because routine fields are just as important, in my opinion, in the early diagnosis of localized intracranial suppuration as a temperature record. (e) Do not fuss about the direction of the nystagmus. Observe each day whether the patient had any spontaneous nystagmus or not. Decide that it is retro-labyrinthine, and use that as a basis. (f) If he has no spontaneous nystagmus ascertain if the vestibular manifestation of increased

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Reference: †Eagleton, W. P.—Cerebrospinal fluid, loc cit, November, 1929, Page 724.

intracranial pressure is present. (g) Do not do a lumbar puncture unless you are sure the abscess does not involve the posterior fossa or the mid brain cone. (h) And do not administer ether when unnecessary if you think you have brain abscess.

If we are to advance we must realize that nobody knows much about this work. To do it well, we should organize teams of two or three men, preferably young men, with good surgical training who have time and inclination to do a work, which is nerve-wracking, and laborious, but most intensely interesting. Such a team in the different eye and ear hospitals would soon develop a diagnostic and operative technic, would rapidly elucidate the factors and problems of intradural suppuration, and save many lives which are now sacrificed because of our imperfect knowledge.

BRAIN ABSCESS FROM THE STANDPOINT OF THE BRAIN SURGEON

WILLIAM J. MIXTER, M.D.

BOSTON, MASSACHUSETTS

The viewpoint of the neurological surgeon in regard to brain abscess must of necessity be somewhat different from that of the ophthalmologist or the otolaryngologist. Fortunately this difference is more superficial than real, for the co-operation between these branches of surgery has been close in the past. It must become even closer in the future if we are to continue to advance in our treatment of this serious disease. To this end we must not only compare our results, we must reconcile our differences and also see and study each other's cases. Finally, we must each of us adopt the advances made by the other, scrapping our own methods wherever we can find better ones.

Let us first consider the allied subjects of pathology and etiology. Brain abscess for the purposes of this discussion may be defined as any localized collection of pus lying within the cranial cavity. (This definition will exclude diffuse inflammatory processes such as encephalitis and meningitis, except in so far as they are associated with true abscess, and will also exclude thrombi except as they are associated with abscess. The pathologic process thus is an abscess lying within the cranial cavity, caused by any one of innumerable micro-organisms or by a mixed infection). The infectious organisms most commonly found would include the streptococcus, staphylococcus, pneumococcus and a mixture of these with other organisms, particularly the colon bacillus. Few surgeons realize how frequently the colon bacillus is found in brain abscess, nor how frequently the pus from a brain abscess may be sterile. These, I believe, represent our usual findings, especially if the abscess is secondary to an infection arising in the realm of the otolaryngologist, but we must be eternally on the watch for multiple or dumbbell abscesses or for strange infections with bizarre clinical pictures and unusual

operative findings. I believe that you gentlemen whose work is primarily devoted to nose and throat diseases see as many or more abscesses than we whose work is primarily neuro-surgery, but that we see more abscesses arising from structures other than the air passages. Among these other infections may be mentioned tuberculous abscess, abscess due to yeasts, and finally those due to fungi such as streptothrix and actinomyces. These fungus infections have forced themselves to my attention during the last few years. A recent experience was of particular interest:

The patient, an elderly man, had had severe right facial pain for several weeks following extraction of some teeth under local anesthesia. The pain came on very promptly following the first attempt at extraction, which was unsuccessful owing to lack of anesthesia following novocaine injection. After some weeks the patient developed an orbital abscess which was opened and drained but did not subside. The process extended gradually with brawny swelling about the eye and exophthalmos, but without elevation of temperature. The next development and the one that brought him under my care was the appearance of signs of increased intracranial tension and right-sided cortical irritation. Operation revealed a small abscess beneath the anterior portion of the temporal muscle, marked thickening and infection of the underlying bone and of the meninges, and an area of porky hardness in the temporal and frontal lobes in which were several small abscesses. Examination of the pus showed actinomycosis. Autopsy revealed that the infection involved the base of the skull apparently entering at the foramen ovale or rotundum. The infection had apparently traveled up the nerve sheaths from the jaw to involve the base of the skull.

Such experiences as the above bring us all to the realization that brain abscess is not a simple lesion but one in which the causative agent may be very varied. Other cases might be quoted as follows:

A child with an influenzal abscess and meningitis following a simple depressed skull fracture; an old gentleman operated on for brain tumor with no thought of abscess, who in reality had a metastatic brain abscess, and a case operated on by my associate, Dr. Hodgson, that showed a tuberculous abscess of the frontal lobe without other demonstrable lesion. Such cases as these are less often seen by the otolaryngologist and seldom by the ophthalmologist, except the case of actinomycosis.

We neuro surgeons must remember, nevertheless, that brain abscess is usually caused by inflammatory disease in the air passages, and that we must look to you to help us wherever this occurs. We must *all* remember that suppuration anywhere in the body may cause brain abscess, though of course suppuration in the lung is probably the most common primary focus after the upper respiratory tract. Blood stream infection is another common cause, but even in the absence of any evidence of septicemia we may have metastatic brain abscess from such distant foci as pelvic abscess and the like.

We must remember that the character of intracranial infection due to adjacent inflammatory disease will vary considerably according to its location. Infection of the frontal sinuses is the commonest cause of osteomyelitis of the frontal bone. This disease is frequently fatal and its course rapid. Should it become chronic or even subacute, frontal lobe abscess is a common complication. Infections of the ethmoid and sphenoid cells, on the contrary, seldom result in abscess. Diffuse meningitis is the rule, and if abscess does occur it is almost invariably a subdural or subarachnoid abscess rather than infection within the brain itself. If we continue from before backward we come to the most common brain abscess, that of the temporal or parietal lobe. Such abscesses arise usually as the result of middle ear disease and it is in these cases that we may find the process occurring by direct extension. I think, however, that abscess from direct extension is far less common than was taught a few years ago and that infection from septic thrombi is more usual. Finally, we come to cerebellar abscess. Acute cerebellar abscess presupposes a coexistent thrombosis as a result of mastoid disease and does not occur by direct extension. Chronic cerbellar abscess presupposes a preexisting sinus infection which may be healed. These facts must be borne in mind in the treatment of these various types of abscess.

When we come to the question of diagnosis of brain abscess we find that we must not depend on our own efforts alone but must call for the co-operation and knowledge of a varied group of consultants of whom the neurologist is the most important.

In many instances the diagnosis is clear with typical history of focus of infection, followed by increasing signs of intracranial extension including headache, somnolence, etc. Examination reveals choked disc, changes in the visual fields, cerebrospinal fluid

changes and evidence of weakness of one side of the body, or cortical irritation. With such a case we have no difficulty, but there are many atypical and puzzling cases that do not present the classical picture of abscess. Take for example the differential diagnosis between brain abscess and brain tumor. These two diseases may present at first glance an almost identical clinical picture. Where, then, may we find important differences? Brain tumor is a non-inflammatory disease, and abscess is inflammatory; yet the temperature chart may be flat normal in both cases. The white count, however, is often a clue. A persistent leuocytosis, particularly with an increase in the polymorphonuclear leucocytes is very suggestive of abscess, and on this alone the diagnosis sometimes hinges.

The cerebrospinal fluid picture is more complicated but is of no less importance in arriving at a differential diagnosis. Pressure relations, chemistry and the cellular elements are all important and for this reason we must insist on a complete and thorough examination of the fluid if lumbar puncture is not contraindicated by evidence of marked increase in intracranial tension and compression of the medulla.

Meningitis is of course easily differentiated by the presence of polymorphonuclear leucocytes and by the decrease in chlorides; hence the diagnosis of abscess with complicating meningitis, as against tumor, is readily made. Even the presence of a small number of polys. is usually a very strong indication of abscess. In both tumor and abscess the pressure is increased, protein is increased, and if there is no meningitis, chlorides are normal. An increase of lymphocytes is strongly suggestive of abscess as against tumor, but not absolutely positive, as we have recently discovered that there are a few tumor cases that show a similar increase in lymphocytes. Thus we must not make a diagnosis of abscess as against brain tumor solely on the presence of lymphocytes. We can say such a diagnosis is probable, but in order to make it certain we must have other presumptive evidence of abscess either in history or the physical examination.

Syphilis and tuberculosis will give a similar cell picture, but these diseases can almost always be differentiated by other means.

Dr. Eagleton has stressed the importance of fundus and visual field examination in abscess. We should take the lesson to heart for here is a diagnostic measure which not only gives us important means of localization, but also gives us perhaps the

optimum time for operation. The differential diagnosis between sinus thrombosis and abscess has been freely discussed of late and is far more in your province than in mine. From the viewpoint of the neurological surgeon, Tobey and Ayers' test for sinus thrombosis by compression separately of each jugular vein would seem to be logical and accurate. In the few cases in which I have seen this test used it has been of definite value, and I know from experience that in tumor this test is not positive. Some confusion may be caused in the rare case where jugular compression causes temporary cessation of the heart beat from stimulation of the vagus. This occurrence is rare and is quickly recognized by the fact that the radical pulse stops. The heart starts beating again as soon as compression is released.

Differentiation between a slowly leaking abscess, particularly a superficial abscess, and meningitis is at times difficult and often impossible. Even an increase in intracranial signs with improvement of the cerebrospinal fluid findings may not tell the story for a certain amount of walling off at the level of the tentorium or below may give the same picture in diffuse meningitis as in abscess. Of course here we are faced with the question—when does walling off of a diffuse meningitis constitute an abscess?

The treatment of brain abscess is essentially surgical but there is nevertheless a good deal of difference of opinion as to how and when. To my mind one of the very important considerations is the original focus, especially if such focus be demonstrated in the nasal sinuses or mastoid. This should be cleaned up as rapidly and completely as possible. Frequently it is wise to do this before attacking the intracranial lesion, particularly if the position of the latter is uncertain or if it is of recent and rapid development.

I am not one of those who believe that a probable case of brain abscess must be explored at once and the abscess drained at all hazards. I believe that as soon as an abscess can be satisfactorily localized a careful but determined effort should be made to evacuate it, but I am firmly of the opinion that many patients have been lost as the result of ill-planned and ill-executed search for a brain abscess before such an abscess had been given time to indicate its position. Better a certain amount of delay rather than a brain, lacerated and infected by too enthusiastic exploration.

During the investigation of the original focus, the surgeon should watch closely for a lead. If one is found he should follow it carefully through skull, meninges and brain and drain the abscess wherever possible through the original focus. For this reason I am firmly of the opinion that abscess of this nature should be handled by an otolaryngologist. There is, however, one very important proviso, namely, that the otolaryngologist be conversant with modern methods in neurosurgery and by training and practice able and willing to apply them. If such an operation is performed by one ignorant of these procedures, the result will probably be deplorable. It is better to wait for a trained consultant than to plunge in ignorance. We at the Massachusetts General Hospital are very fortunate in our close affiliation with the Eye and Ear Infirmary and the close proximity of the two institutions. We are often able to combine our forces to good advantage, and have occasionally been able to carry out the following plan. The exploration of the original focus and, if possible, drainage of the abscess is carried out by the otolaryngologist. Then if a free exploration of the brain through a clean field is indicated, the neurosurgeon, who has been present from the beginning, can step in with a complete fresh layout and carry on without loss of time. In many institutions such cooperation is not possible. Under these circumstances the surgeon, be he otologist or neurosurgeon, must be prepared to carry out both procedures. On account of the special training and technique necessary for such operations as exploration of ethmoids, sphenoids or mastoid, the otolaryngologist should usually have the preference if the operation is to be carried out entirely by one man.

We have already spoken of the etiology of abscess, particularly the fact that some of them are caused by inflammatory processes in the nasal sinuses or mastoids, and others are frankly metastatic. We have also spoken of the advantages of exploration through the original focus if this is possible. Here is a word of warning. The neurosurgeon must take care not to overlook the possibilities of direct extension and the otolaryngologist must investigate his cases from all angles that he may not overlook some distant primary focus.

If a brain abscess has been located through the original focus we must assure ourselves that drainage is adequate before we stop. In many parts of the body partial drainage of an abscess cavity may, effect a cure, but this is not true in the brain. There

are many factors that contribute to this, and among them may be mentioned the formation of a dense wall of inflammatory tissue about the chronic abscess, the lack of power of resistance and repair in brain tissue, and the well known tendency of brain abscess to burrow deeper toward the ventricle rather than toward the surface. Thus it is frequently necessary to drain the abscess *de novo* through a clean area even if it has occurred by direct extension. Drainage through a clean area is almost always necessary in subcortical metastatic abscesses.

In this matter of exploration and drainage through a clean area we find that both the otolaryngologists and the neuro surgeons have contributed to our knowledge. To men like Cushing and Frazier we owe the technique of modern brain surgery, but the more recent advances in exploration and in the technique of drainage we owe to Eagleton, Mosher, King and others. Cerebral abscess can be searched for in one of two ways, either through a small drill hole or a good sized bone flap. The choice of type of operation depends somewhat on the definiteness of the localizing signs, condition of the patient and location of the abscess. I personally have a leaning toward exploration through a drill hole, particularly if the patient is in poor condition. A small nick only is made in the dura and the exploration is made with a large, round-pointed Cushing needle. Great care should be taken not to puncture the ventricle. (Punctures are made in various directions and to various depths.) If the abscess is reached at an angle favorable for drainage, two courses are open —one to open and drain it completely at one sitting, and the other to do it in two stages as in a lung abscess. If the latter course is decided upon only as much pus is evacuated as is necessary to relieve extreme tension, and the needle withdrawn. The opening in the skull is then enlarged, the dura turned back and the exposed brain covered with cotton. The scalp is closed tightly over the cotton and left untouched for twenty-four hours, thus permitting a certain amount of walling off before the abscess is freely opened. At the second stage which can usually be carried out without any anesthetic, the abscess is again opened and treated according to the desire of the operator, either by resection of the overlying brain, insertion of a Mosher drain, or simple drainage with a rubber tube. One must be guided in one's choice by the depth and position of the cavity and by the thickness of its wall. I believe that where it is applicable, the King operation

should be of great value though my experience with it has been small. I do not believe that one should resect the motor cortex, speech or visual centers, or brain in the vicinity of these structures in order to apply this procedure. If the surgeon prefers to handle an abscess at one sitting rather than two, the same procedure can be carried out or a bone flap turned down if desired. The one outstanding disadvantage of the two-stage operation is the possibility of losing the abscess and not finding it again. This I believe is more than outweighed by the lessened risk of meningitis if this procedure is carried out.

We should remember that brain surgery demands a careful and delicate technique—one that differs markedly from any other kind of surgery and that we must apply this technique in our handling of brain abscess. Careful and accurate hemostasis is essential, and this cannot be obtained by hemostat and ligature alone. The use of Cushing clips, cotton pledgets and bits of muscle should be familiar to us all. More recently has come the use of the Bovee apparatus, or some modification of it, whereby cutting and hemostasis are obtained by means of electro-coagulation or dessication. Such an apparatus is a great help in all brain surgery and almost an essential for excision of brain tissue, as in the King operation. I have spoken of the use of a Cushing ventricle needle to explore for abscess. For such use the hub of the needle should be bored to take a syringe point or, better, a metal adapter fitted to a rubber tube. If this is used pus need not be spilled in the wound. It is essential to keep the needle in place after the abscess has been reached until the opening has been sufficiently enlarged and the drain inserted. The drain should project well into the abscess cavity and be held in place by sutures in the scalp. Personally, I have found the Mosher drain more satisfactory than any rubber tube or combination of tubes. Drainage of acute cerebellar abscess with the usually coexisting sinus infection is very definitely in the province of the otolaryngologist. Chronic cerebellar abscess should be drained through a clean field.

Everyone must use his own judgment in the after care of brain abscess, and the process varies so much that each case is more or less a law unto itself. The essentials are that drainage shall be complete, that the drain shall remain undisturbed for a long time, weeks if necessary, and that the back wall of the abscess be left intact. I have never made any attempt to sterilize

the abscess cavity. If there is any possibility of extrusion of the wall of a thick-walled abscess, it should be encouraged, but no attempt should be made at rapid removal. If there is a complicating meningitis, aseptic or septic, it should be treated by repeated lumbar or cisternal puncture.

The prognosis of brain abscess depends on the virulence of the organism, the position of the abscess, and the presence or absence of a so-called capsule. It is true that the early soft-walled abscess is difficult to treat and the results are not good. I do believe that there is little to be gained by precipitate operation on a septic infarct in the hope that it may be an abscess.

The neurosurgeon can bring you nothing new and startling in the diagnosis of brain abscess. He can perhaps offer you certain refinements in the technique of brain operations which are not needed in operations on the nose, throat and ear. He must, however, call on you for assistance in the care of the original focus in most cases of brain abscess, and it is only through the continuation and extension of the cooperation which has existed between the otolaryngologist, the ophthalmologist, and the neurosurgeon that we may further benefit our patients and increase medical knowledge.

BRAIN ABSCESS—METHODS OF DRAINAGE

HARRY P. CAHILL, M.D.

BOSTON, MASS.

Mr. Chairman, Members of the American Academy of Ophthalmology and Otolaryngology:

It is a bold task to address you on the subject of brain abscess when one is not bringing forward anything new or original. Were it not for the fact that I feel so earnestly the advantages of the transmastoid and subtemporal approaches in most cases of otitic brain abscess, I should hesitate to advance my views on this subject for your consideration.

Results in the treatment of brain abscess will vary greatly with the nature of the condition. No definite method will be suited to every case. Any procedure will demand a complicated technic. Any operation will be dangerously traumatic. The simpler we can make its execution, the more perfectly it will be carried out, and the evils of hemorrhage, oedema, and laceration of tissue will be greatly lessened.

This technic of necessity embraces two salient points: First, the avenue of approach; and second, the method of drainage. The simpler we can make this approach, the simpler our type of drainage, giving due consideration to the problem before us, the better our results must be.

In abscess of the temporo-sphenoidal lobe, the approach must be either through a bone flap, a temporo-parietal decompression, a subtemporal decompression or through the mastoid tegmen extended. Approach by means of the bone flap gives a large field of exposure, some advantages if a certain type of meningeal abscess is present, and less danger of infection if the abscess is not located. But it does add to the operative time; it does add to the possibility of trauma. Hemorrhage from the postero-inferior border of the bone flap may be exceedingly troublesome. The abscess, if localized, is given opportunity to invade freshly opened tissue.

Accidentally, even the infected aural area may enter the field. Theoretically, at times it has advantages; practically, in the hands of the otologist this is not so.

The subtemporal decompression has many of the favorable points present in the bone flap. In moribund cases with a sclerotic mastoid, not yet operated upon, it is the approach of choice. When the mastoid is of a type requiring the use of a mallet and gouge, or the technic of the operator includes it as a necessity, this operation is of value.

Approach through the mastoid or tympanic tegmen has given a higher average of recoveries than any other method; the meningeal approach and even the cerebral approach is often walled off. Here granulations on the dura or exposed dura guide one to the meningeal or intracerebral abscess in over fifty per cent of cases. Here, the abscess is frequently nearer the cortex. Here, a fistulous stalk opens at times. Here, the cortical cells do not belong to any important cerebral centre. In meningeal and cortical abscess, and where the abscess is not localized, its disadvantages can be reduced by remembering these possibilities in the operative technic.

The ideal treatment of a brain abscess would be to remove it *in toto*. This has been successfully accomplished; but the surgical shock, the encephalitis, the hemorrhage and danger of infection from the stalk, if present, or from the diagnostic puncture, or from rupture of the capsule, are insurmountable obstacles in the way of its success.

Partial removal of the wall of the abscess by unroofing it, with herniation, greatly decreases the dangers of the removal *in toto*. You are all familiar with the essentials of the King operation. It is one of the brilliant achievements in American surgery. The danger of recurrence due to partial collapse of the capsular wall of the abscess cavity or to retained infection and secretion of the capsular wall, is reduced to a minimum. The secretory capsular lining, whatever little remains, is replaced by granulation tissue. Drainage is not a serious problem.

In frontal lobe and in temporo-sphenoidal abscess, especially if it is on the right side, the King technic must be given ample consideration. In cases unsuccessfully drained through the mastoid, it has taken care of the recurrence.

The other methods of treatment vary from complete evacuation and inspection of the lining capsule through the encephaloscope to simple repeated punctures.

The type of drainage may range from the fenestrated metal or glass cylinder and wire gauze to rubber tubing and tissue. We must be open-minded and let the emergency guide our choice. All drainage is for the purpose of emptying the abscess cavity so that the intracranial pressure may obliterate it. Once the drainage tube is in place, various aids may be employed to assist the cerebral pressure, such as rotation of the head, gravity, lowering of the top of the table, suction, etc.

The removal of a cone of cerebral tissue reaching to the capsular wall by means of the radio knife and loop of Bovie has proved very satisfactory in several recent cases. It has permitted the wire gauze drain to be placed more accurately and with less trauma and hemorrhage. Hence the use of the larger Mosher drain will become more common.

The formation of a fistulous tract about the drain to the abscess cavity in part makes the capsule extend to the surface of the brain. This approaches in a degree the unroofing method of King. Hence the drain, when in proper position, should not, ordinarily, be disturbed for from four to six weeks. It takes a considerable time for neuroglia tissue to form about the tube. After that time, the stay sutures may be incised and the brain permitted to expel the drain slowly.

Lipiodol injection has been of value in several of our cases. Its use is indicated in all drainage cases with persistent purulent discharge, headache, or other questionable signs or symptoms. It has made clear in some cases that the drain was not in proper position and was outside the capsular wall. It has shown how easily recurrence can be favored by imperfect collapse of the wall. It has been, in a way, a measure of the abscess drainage.

There is in the school of the present day one group favoring larger and larger drainage openings, while another is using repeated puncture and suction. Rubber drains of fine calibre are favored by the latter group.

In the treatment of cerebellar abscess as in abscess of the middle fossa, there is some question as to the avenue of approach. If symptoms or signs of labyrinthine involvement preceded those of cerebellar infection, the paramastoid route through Trautmann's triangle, with at times obliteration of the lateral sinus, is

the route of choice. When the abscess is secondary to sinus involvement, drainage through the inner sinus wall has been our customary procedure. If exploration of the mastoid cells posterior to the sinus reveals the presence of an extra-dural abscess, the area of protective meningeal adhesions should be used.

The importance of deciding which of these three avenues has a given area of meningeal protection may mean the difference between success and meningitis. Like so many of our operative points, we are indebted largely to Dr. Eagleton for this. The wisdom of bilateral occipital decompression in such cases is better left to the neurological surgeon.

The importance of measuring the spinal pressure in temporo-sphenoidal abscess, and of the lateral ventricle of the unaffected side in cerebellar cases immediately before the operation, must not be overlooked.

In conclusion, the mistakes one may make in certain intra piarachnoid, cortical and unlocalized abscesses will be more than compensated for by the value of the protecting meningeal adhesions and cortical reaction over the avenue of infection from the mastoid. The radio knife and lipiodol injection have proved valuable. The simplicity of this technic with the Mosher drain will appeal to the otologist who does not possess the facilities nor the opportunity for keeping his neurological surgery at its best.

DISCUSSION OF SYMPOSIUM ON BRAIN ABSCESS

DR. JOSEPH E. J. KING, New York City: Mr. Chairman: I thank you for the very courteous invitation to take part in the discussion of these most important papers, all of which I have thoroughly enjoyed.

I first want to take an adverse stand in regard to Dr. Eagleton's remarks relative to his own power of "observation," for if one reads his book, which he says is full of errors, one will find the most minute descriptions of pathology, just what one should see if he looked. His description of pathology in his book makes one feel very much as if he had attended the autopsy himself.

I shall confine my remarks to the treatment of brain abscess. The majority of cases with which we have had to deal were cerebral rather than cerebellar abscesses. Regarding the prognosis, I do not think there is very much difference between the results obtained in temporo-sphenoidal and frontal abscesses. So far as the type of infection is concerned it has been my experience that in those in which the streptococcus is present in encapsulated abscesses, the results are more favorable. Next comes the staphylococcus, and the least favorable is the pneumococcus.

We have all seen cases of suspected brain abscess in which we did not know whether an abscess was present or not. In all such cases it is impor-

tant to get rid of the source of infection first—except in the case of a metastatic abscess—either by a proper mastoid procedure, or a proper operation for ethmoid or frontal sinus infection. Much to my sorrow I operated upon a man for frontal lobe abscess in which the abscess was not present. He had frontal sinus disease, with an extradural abscess, and previous to the operation on his frontal sinus I made an unnecessary exploration in the frontal lobe. I had two cases which should have recovered, but which died, the one in which the frontal sinus operation was not done until too late, and the other in which the operation on the frontal sinus had previously been done three months before, but in which reinfection took place. After the brain abscess was practically well she developed a secondary brain abscess from the frontal sinus. In cases of doubt it is my opinion that one should first attack the source of infection rather than exploring the brain and looking for something which may not exist.

From the practical standpoint all we care about in any of these operations or procedures is that the patient lives. We do not care whether he survives as a result of one procedure or another. The procedure which gives the best results in the hands of the operator should be used. No one procedure will cure all brain abscesses, and I do not think that all combined will do so. There are certain cases in which the Mosher drain is probably the most logical one to use. There are some cases in which a small drain, especially in the deep-seated abscess, will probably do much better. Such a drain has been used by Dr. Coleman of Richmond; and there are cases in which the procedure which I have suggested will probably give the best results. I do not think that any one type of operation will give good results in all cases. Therefore, we should be prepared to use the type of procedure applicable to the case in hand. Dr. Eagleton has used an inverted osteoplastic flap. Dr. Adson has used an osteoplastic flap with the drain brought out through its central portion. Dr. Dandy proposed a tapping of the abscess without aspiration to relieve the pressure, and let it go at that. Some claim that the contents of the abscess will become less and less virulent, and may even become absorbed, and state that the patient gets well. I do not know about this type of procedure. Many of these patients undoubtedly go home and are well for some time, but I am under the impression that it is not logical to let a patient go around with such a potential volcano in his head. Dr. Coleman has used a small drain which is left in position in the cavity for a long time, and it is surprising to know the results which he has obtained. In the group of cases which he reported to me there are included 20 cerebral and 4 cerebellar abscesses. Metastatic abscesses were not included. Of the cerebral abscesses, three died, two of meningitis before operation. Of the cerebellar abscesses two died. This is indeed an extraordinary group of cases.

I have advised herniation of those abscess cavities which are suitable for such a procedure after one case in 1920 which accidentally herniated and recovered. Other cases which had been drained with rubber tubes have died. After three cases had recovered by allowing the abscess cavity to herniate, I was advised by Dr. Berry to write a paper on the subject. The procedure which I advised was described in this paper. In these

cases and subsequent ones herniation of the abscess cavity outside of the cranial cavity was desired and accomplished. These, and others later in which a similar procedure was done, recovered. I would not use this procedure in all cases. One cannot expect an abscess at the base of the brain to be cured by herniation. I think probably I have made two mistakes in the original procedure. In the first place, I think the opening in the skull should be somewhat smaller. In the second place too much herniation should not be allowed. Practically all of the cases which I have had that recovered were encapsulated. When one inserts the brain canula for exploratory purposes and the "capsule" of the abscess is located, there is a distinct resistance offered to the canula producing a certain sensation or feeling—similar to the resistance offered by a thin-walled rubber ball in mush. So, instead of making such a large opening in the skull and opening the abscess immediately, in the next case which I operate upon I propose to remove the overlying brain tissue and allow the abscess which has not yet been punctured to herniate somewhat, so that adhesions may form about it previous to opening on the following day, thus preventing meningitis. This suggestion is similar to that of Dr. Mixter. At the end of 24 hours a certain amount of herniation will have taken place, and adhesions will have formed between the brain cortex and the dura so that there is practically no danger of spreading the infection. Opening the abscess on the following day is nothing more than a dressing. The overlying brain tissue is of but very little value. This tissue bleeds but little when removed. Most of the cases with which we have to deal are in the temporo-sphenoidal or frontal regions, and but few in the parietal, so I doubt if it makes any difference whether this brain tissue which has lost its blood supply is removed or not.

While coming here yesterday I considered the question of over-herniation of the abscess cavity, and thought that perhaps in some of my cases too much tissue was allowed to herniate. I was considering the idea of having some device made of similar material to that used in Dr. Mosher's drain, but rather the shape of an old cotton basket than like a cornucopia, of different sizes to fit the different sized abscesses. I considered inserting this type of basket-like drain into the abscess cavity, allow the cavity to herniate until it was on a level with the skull, then remove the device and prevent further herniation. Once the cavity has herniated until none of it remains in the skull, the object of herniation has been accomplished. From this point onward, the area should be allowed to granulate over and heal. This I propose to do with the next case of encapsulated abscess upon which I operate.

I have had seven cases which got well, and I have seen two cases die which should not have died. I have had several cases, upon which I operated, die, but in these cases no hopes for recovery existed. I want to reiterate the importance of late operation for brain abscess. Some patients may die by waiting, but there is no doubt that when encapsulation has taken place the prognosis is more favorable. In all cases in which encapsulation has taken place the patient has passed through a stormy period at first, and finally encapsulation took place. If we could only keep our fingers off, and not allow ourselves to be rushed into a situation due to

the family wanting us to do something for the patient, and if we could wait long enough to allow encapsulation to take place, I think we will save many more of these patients. Only one of my cases which recovered was non-encapsulated.

Several years ago brain abscess by the profession in general was considered a hopeless situation as most cases did not recover. Therefore interest lagged. In recent years, however, more and more papers have been written on the subject, and everybody has become more interested in it. So if at such meetings as these everyone tells everyone else what they think about it, and discusses the subject, eventually we will have improved methods and procedures, and in the end we will be able to lower the mortality rate in brain abscess.

Dr. S. J. Kopetzky, New York, N. Y.: After the excellent symposium presented this morning little remains for me but to summarize, to stress some of the salient points and mention some of the outstanding facts.

The standpoint of the ophthalmologist, of the surgeon, and of the otolaryngologist have been presented to you. Let us look at the question from the standpoint of the clinician. As one sits amazed and stunned, listening to Dr. Eagleton, one realizes that diagnosis is still an art and cannot be done by any set rules or definite formula. And on that art hinges the handling of brain abscess. As Dr. King said, all the procedures combined and applied to certain cases would not cure them. We should first separate the cases in which we can reasonably expect a cure from those in which no method that we have on hand can produce a cure; and we should not operate on the latter cases. Such operations only throw surgical procedures into disrepute.

Dr. Eagleton stressed the history. We should know the pathology that preceded the suspected brain abscess, know it very definitely. Abscesses are either capsulated or non-capsulated, and I partly disagree with Dr. King that in all abscesses you should wait indefinitely for encapsulation. Abscesses that are of hemorrhagic origin, or come from infarcts into the brain, move too fast the lesion develops so quietly that the patient is soon dead—before any evidence is presented that we can see clinically. So while it is proper to wait for rational development and not rush in during the very early period still we must realize that certain abscesses will never form a capsule, especially in cases where the abscesses are multiple.

I wish there was agreement among the ophthalmologists. I wish the reports that we get were written in common terms that all could understand. One hears quite a distinguished member of this society say that there is considerable confusion as to what a given man means by optic neuritis and swelling of the nerve head. That ought to be straightened out. We are not speaking the same language and it leaves us at a disadvantage.

The ear presents the definite lesion. The chronic cases produce an abscess of one type, and the acute cases another, according to the lesion in the ear. My own experience is that the average type produces an abscess close to the ear. With the hemorrhagic type of mastoid—the septic type—the lesion in the brain may be anywhere.

The neurologist's standpoint was not presented this morning. My experience with these gentlemen is that they are so impressed with the slow growth of tumors that as a class they do not appreciate the more rapid growth of abscesses and the swifter change in the picture in these cases. Usually the otologist will make the diagnosis before the neurologists have summarized the varied reflexes or absence of function in the nerves they study. We must learn to ascribe greater significance to minute changes in the nerve-reactions, so as to get an earlier diagnosis localizing the given lesion.

There is much in what Dr. Eagleton says, and I would be the last to deny it. But men of Dr. Eagleton's genius are beyond the rules and regulations that govern the rest of us. Many of the observations he makes and speaks about, I hope later to be able to place in their proper category and use. I trust his observations, but I fear I am unable to follow his reasoning. The fault may be mine.

We are dealing this morning with abscesses and not with infarcts. That is an entirely different lesion. An infarct does not necessarily result in an abscess.

I would like to make one point. That is, that I thoroughly agree with Dr. Mixter that lunbar puncture can be done if performed as he describes it, in almost every case. It should not be done roughly and uncontrolled by a manometer.

It is hard to do justice to a discussion such as this in the time permitted, but I thank the officers for the courtesy extended to me to add what I can to the discussion of this difficult subject.

OPHTHALMOLOGIC SESSIONS

VISUAL FIELD STUDIES

Marked Concentric Contraction of the Visual Fields—A Study of 100 Consecutive Cases

WILLIAM D. ROWLAND, M. D.,
AND
ALLAN WINTER ROWE, Ph. D.

BOSTON, MASS.

The present paper deals with the results of an analysis of the eye examinations on a series of nineteen hundred (1900) patients who were seen in the course of a general diagnostic service. No selection in these cases was made by the opththalmologist, the eye examination being a routine procedure on all House patients on this service, while additional data came from a complementary out-patient service where the selection of cases for the eye examination rested in the hands of the examining physician with whom each patient first established contact.

In so large a number of examinations made under such conditions a number of departures from the normal will present for which no patent explanation is directly available. A series of cases, such as those under consideration forms a concrete illustration. Analysis of all of the available data performed on the patient may bring to light, however, through a series of unmistakable correlations, suggestions as to etiological significance for the presenting condition.

Among the nineteen hundred (1900) cases constituting this series of cases, there appeared from time to time individuals presenting marked contraction of the form and color fields, usually concentric and in a large percentage symmetrical. Few of these patients disclosed the usually recognized causes of such a phenomenon, and the series was allowed to grow until enough cases were available to render a statistical analysis suggestive. As a matter of fact there were one hundred and two (102) individuals in this series of nineteen hundred who presented marked concentric contraction, and the present thesis is a discussion of

the probable etiology of the condition as drawn from the data of the extensive diagnostic study. That certain of the statistical observations shall have meaning the eye examinations here recorded constitute a consecutive series. All of the patients resorting to the Clinic did not receive eye examinations for reasons that need not be discussed here. For this reason the total number of patients examined constitutes a definitely larger group, and an analysis of the relationship of total incidence of disease entity to the eye examination will serve to illustrate the representative character of the complete series. The significant figures are collected in Table I.

TABLE I

CLASSIFICATION OF CASES

	Total	With Eye Examination	
Endocrine			
Pituitary	663	565	85%
Thyroid	400	234	59%
Gonad	309	188	61%
Adrenal	13	13	100%
Pancreas	10	8	80%
Pluriglandular	19	18	95%
Not Endocrine	1031		
Additional			
Repeats	229	874	70%*
Unclassified	223		
Incomplete	93		

*Omit "Repeats" and "Incomplete."

As will be noted in the above table, some fourteen hundred (1400) of the cases presented a definite endocrinopathy as the primary cause of their disability; another thousand (1000) were equally free from any evidence of endocrine disease while demonstrating a definite etiology unassociated with the ductless glands. Again that the representative character of this group may be patent the following enumeration of groups of cases can be given. Patients presented tuberculosis, atrophic rhinitis, arthritis, cholecystitis, various primary focal infections, neuroses, psychoneuroses, psychoses, a great variety of primary lesions of the central nervous system, epilepsy, the Parkinsonian syndrome, chorea; in addition, patients presenting mental arrest, malnutrition, obesity, gout, Paget's disease, primary anaemia, the leukaemias, haemophilia, malignant tumors, benign tumors including a group of non-toxic goiters, syphilis, and asthma. In addition to the above, groups of patients had various diseases of the heart,

the kidneys, the eye, ear, skin, liver, gastro-intestinal tract, and pelvis. It is understood that none of the individuals in the above list gave any evidence of a concomitant endocrinopathy.

Reverting to Table I, the repeat group represents patients who had already been studied and who returned for examination to ascertain the effectivity of the various therapeutic measures which had been followed, and the incomplete group comprised the small number of individuals who for a variety of reasons failed to carry through all of the details of the initial study. The eye examinations of these two groups are omitted from the general compilation. The unclassified fraction derive chiefly from the out-patient service and was composed of individuals who had completed the original investigation but whose etiological classification could not be determined with certainty. As has already been pointed out by one of us[2] the larger part of the cases of this group would have been susceptible to diagnostic resolution had opportunity for additional study presented. The eye examinations in this group, however, constitute a part of the routine series and in the compilation of the relative data they are bracketed with the non-endocrine group for purposes of convenience.

It will be noted that the lowest percentage is shown by the thyroid and gonad cases. In the first instance, the other diagnostic observations many times were so definitely suggestive as to render an eye examination superfluous, while the gonad group—composed exclusively of ovarian failure of functional or surgical origin—offers the poorest measure of individual cooperation in the detailed and extensive diagnostic studies on which these data are based. In every case, however, it may be said that a representative portion of the individual group had the routine eye examination which constitutes the basis of the present thesis.

In this series, then, of nineteen hundred (1900) consecutive eye examinations, a certain number of patients were observed who presented a symmetrical contraction of significant proportions of one or both visual fields. The recognized causes of this phenomenon in the past have been glaucoma, optic and retinal atrophy, hysteria, and certain brain lesions while a concentric contraction with unequal defects has been usually referred to an optic neuritis.[8 9 10] When the first of these fields was observed and evidence of the several factors noted above proven to be absent, the case was filed for reference until such time as added

information might throw light on the causation of the phenomenon. With the completion of one hundred cases showing marked concentric contraction and demonstrably free from the recognized causes noted above, the authors have felt that a careful analysis of the existing data might throw light on the causative mechanism or mechanisms. As a matter of fact the series contains records of one hundred and two (102) cases, but as two of these are in the unclassified group, they are noted only in the first discussion and are then eliminated from the further analysis.

In Table II is given the primary etiological classification, together with certain other pertinent data. In reviewing the data on these patients it must be borne in mind that the primary thesis is to ascertain, from statistical analysis if possible, disease trends which may be regarded as of possible causal significance.

TABLE II

PRIMARY CLASSIFICATION

Diagnosis	Total Number Examined	Contracted Fields: Total + No.	%	Uncomplicated No.	%	Complicated No.	%
Pituitary	565	31	5.5	18	58	13	42
Thyroid	234	12	5.1	2	17	10	83
Gonad	188	6	3.2	1	17	5	83
Lesion Central Nervous System	100	21	21.0	18	86	3	14
Syphilis	47	9	19.1	7	78	2	12
Tuberculosis	29	13	44.8	7	54	6	46
Liver	77	6	7.8	2	33	4	67
Focal Infection	28	2	7.1	2	100	0	0
Unclassified	223	2*	0.9				

*1. Silicosis (potential TB, familial.)
*2. Amenorrhoea (study incomplete.)

Inspection of the table reveals the following facts:

Practically half of the patients (49) present a primary endocrinopathy. Of the pituitary and thyroid cases, between 5% and 6% of those examined showed contracted fields; while in the ovarian group the incidence was but 3.2%. In the non-endocrine series, however, 21% of those exhibiting lesions of the central nervous system exhibited the anomaly, while more than double that percentage of those cases with tuberculosis showed the phenomenon. The percentage in the groups with hepatic disease and with focal infections are the only two of the non-

endocrine fraction approaching the relative incidence in the nedocrine cases.

A word may be said at this point in regard to the two unclassified cases as subsequently they will be dropped from the further discussion. The first was a young man (See protocol Case S-958 at end of paper), who presented marked field contraction but gave no evidence of endocrine disorder. He had been a quarryman and X-Ray of the chest showed the existence of a silicosis. That this condition predisposes to tuberculosis has been suggested and there can be no question but that it involves a very definite disease condition of the lungs. The case is doubly interesting as the young man's father showed the same condition (See protocol Case S-957 at end of paper) but himself gave a definite history of serious head trauma. This familial incidence will be noted elsewhere in the body of the paper.

The second case was a young woman presenting amenorrhea as her chief complaint. A complete study could not be secured and for this reason the underlying disease condition not properly evaluated. It is possible, however, that hysteria was a factor in the case which would exclude it from further consideration even if it were not unclassified. As the diagnostic studies were as complete as possible and involved both a thorough clinical investigation and a long series of quantative laboratory measurements, and since, further, endocrine diagnosis were established only after the elimination of non-endocrine possibilities by objective test and observation, it follows that a certain percentage of the cases presenting demonstrated the presence of a secondary complication of potential import. An analysis of the hundred cases forming the basis of this discussion from this standpoint discloses a number of highly interesting facts. Reference to the table will show that 58% of the pituitary cases were without significant complications that only 17% each of the two remaining endocrinopathies exhibited that condition as the single cause. Furthermore, the complications in all of these groups fell under the several non-endocrine entities listed in the table.

86% of the cases primarily presenting a lesion of the central nervous system were uncomplicated; three fourths of the syphilitics, one-half of the tuberculous patients, and but one-third of the hepatic group. From the raw data of this table one might well draw the inference that lesions of the central nervous system and syphilis were patently associated with the eye condition, that

pituitary disease and tuberculosis were possible etiologic factors, and that thyroid and ovarian disease and hepatic dysfunction were not of primary importance and of a doubtful causality. A further analysis, however, of the raw data permits of a more exact interpretation providing as it does the factors from both the primary and the secondary disease conditions.

TABLE IIa

TOTAL CLASSIFICATION

Diagnosis	Primary Est.	Primary Probable	Primary Total	Secondary Est.	Secondary Probable	Secondary Total	Grand Total
Pituitary	30	1	31	0	0	0	31
Thyroid	10	2	12	0	0	0	12
Gonad	6	0	6	1	0	1	7
Lesions Central Nervous System	11	10	21	10	7	17	38
Syphilis	6	3	9	1	4	5	14
Tuberculosis	8	5	13	7	6	13	26
Liver	5	1	6	9	3	12	18
Focal Infection	2	0	2	9	0	9	11
	78	22	100	37	20	57	157

In assembling the data for this table distinction has been made between the established diagnosis resting upon a definite objective basis and the probable incidence of a disease condition in which past history, circumstantial evidence and other less objective factors are adduced for support or establishment. It will be noted, for example, that one of the pituitary cases is regarded as probable, while thirty are established. Two of the thyroid cases, however, out of twelve must be regarded as probable inasmuch as a superimposed non-endocrine condition influenced certain of the important objective findings, and while allowance can be made for this influence, there remains some element of assumption in the primary diagnosis. In fairness, however, it should be said that in the endocrine cases therapeutic tests have supported the validity of the initial diagnosis. With the syphilitics, the tuberculous, and those patients presenting involvements of the central nervous system, subjective evidences play a larger part. Over three-fourths of the cases, however, are classified on the basis of definite establishment, while the remainder possess that degree of probability which would constitute, under ordinary conditions, a well-founded working hypothe-

sis. With the secondary factors the established group contains twice as many patients as does the probable fraction. That there may be no misunderstanding it should be emphasized that in the "probable" group there is in every case a mass of supporting evidence but the total picture lacks somewhat of the completeness of the better authenticated section.

Analysis shows here one endocrine case with an endocrine factor as a secondary element. This was the single patient in the series presenting a so-called pluriglandular syndrome, which in her case consisted of a dysfunctional state of the pituitary superimposed upon a hypogonadism deriving from an earlier surgical castration. In the interest of simplicity this patient has been listed as a pituitary case with the ovarian factor as a complication. It will be seen from the table that the one hundred cases of the series present, one hundred and fifty-seven (157) diagnostic elements; while reference to Table II will show that the additional complications, fifty-seven (57) in number are divided among forty-three (43) cases. In other words, several of these patients present evidences of two or more of the conditions which are seemingly involved in the study.

A further resolution of the distribution of the complicating factors throws more light on the general situation. In compiling these, focal infections are included, as two of the patients presented no other demonstrable pathology than that single condition. We are inclined to believe, however, from a survey of the many hundreds of cases presenting focal infection as such as primary or complicating factor that it does not assume the same degree of importance as do the other four non-endocrine groups. An analysis of the complications is given in Table III.

TABLE III

Primary	Uncomplicated	Complicated					
		Gonad	Central Nervous Lesion	Syphilis	Tuberculosis	Liver	Focal Infection
Pituitary	18	1	4	2	5	5	5
Thyroid	2	0	2	0	3	5	0
Gonad	1	0	3	0	2	1	2
Lesion Central Nervous System	18	0	0	0	2	1	0
Syphilis	7	0	1	0	1	0	0
Tuberculosis	7	0	4	2	0	0	2
Liver	2	0	3	1	0	0	0
Focal Infection	2	0	0	0	0	0	0

Inspection of the table shows that while the larger part of the pituitary cases are uncomplicated, the remaining thirteen show several secondary involvements. The predominence of liver cases in the thyroid group emphasizes once more the frequent association of hepatic dysfunction with thyroid failure that has already been reported by one of us.[3] Similarly this datum bears on the liver figures, emphasizing that a real factor in its relatively low incidence is the complication of endocrine disease. The majority of the complicated primary liver conditions show some involvement of the central nervous system as the second factor. The association of lenticular degeneration with jaundice may be no more than coincidence in connection with these data. It offers, however, a suggestive lead for future investigation.

The relative incidence of each factor, irrespective of primary or secondary character, may be given most compactly in tabular form.

TABLE IIIa

Diagnosis	Primary and Secondary No.	%	
Pituitary	31	19.7	Total Endocrine 31.8%
Thyroid	12	7.6	
Gonad	7	4.5	
Lesion Central Nervous System	38	24.2	Total Non Endocrine 68.2%
Syphilis	14	8.9	
Tuberculosis	26	16.6	
Liver	18	11.5	
Focal Infection	11	7.0	

Lesions of the central nervous system are present in nearly one-fourth, and pituitary disease in nearly one-fifth of the total group. Tuberculosis also is an evident factor, and over one in ten show some degree of hepatic involvement. It is, of course, possible that the figures for the syphilitics derive from those in whom the disease has attacked the central nervous system, though it will be noted in the analysis in Table III that but one of the nine cases gave evidence of central neural involvement. The really striking thing from all of these tabulations is the definitely limited number of disease states which present this peculiar condition out of the wide variety examined. Furthermore, it can hardly be fortuitous that in the endocrine group manifesting the

condition, only 17% respectively of thyroid and ovarian disease (3 cases) failed to show a non-endocrine involvement identical with one or another of the non-endocrine conditions which constitute half of the presenting group. The anatomical relationship of the pituitary gland with the possibility of traumatic injury to adjacent structures as a result of its enlargement, introduces what we may call a mechanical element in its influence which is sharply borne out by the statistics as compiled. In other words, if we include the somewhat dubious luetic factor, over 50% of the group present conditions which may be regarded as involving directly or indirectly the central nervous system.

An analysis of the twenty-one (21) cases in which a disorder of the central nervous system is the primary factor may not be uninformative. The data are collected in Table IV.

TABLE IV

Analysis of Group of Central Nervous Disorders.

Parkinson Syndrome	1
Little's Disease	1
Paget's Disease (with pressure)	1
Epilepsy (with degenerative change)	2
Brain Tumor	1
Meningitis	1
Head Trauma	6
Mental Defective	3
Hydrocephalus	2
Cerebral Hæmorrhage	1
Arterio-sclerosis	2
Total	21

Review of the table indicates the protean character of the conditions involved and defines the impossibility, at this time, at least, of a more detailed analysis of the causal relationship with the presenting symptom. The head trauma cases, the largest single group, all gave histories of severe injury with presumptive damage to the central nervous system. The three mental defectives were possibly all cases of birth injury. The other items require no elaboration.

In this group there were a number of individuals who have been classed as mentally defective. In the majority of instances this was a trend rather than an established condition, as in many mental defectives it is patently impossible to secure that meas-

ure of cooperation necessary for the eye examination. Each of the patients in the series who showed any mental inferiority was re-tested a sufficient number of times so that the correlation of the eye data offered an authority to the final form. In the entire series of three thousand (3,000) cases there were children presenting obviously impaired vision associated with defective mentality. Attempts were made with all of these children to secure eye studies. Where, however, the mentality was so low as to preclude authoritative measurements, they have been dropped from the series. The allocation of patients with mental defect found in the next table shows a relative uniformity of incidence with the exception of the central nervous system group and even more strikingly that of the syphilitics.

TABLE IVa

Diagnosis	No.	Defective (Mental)	%
Pituitary	31	5	16
Thyroid	12	2	17
Gonad	6	1	17
Lesion Central Nervous System	21	5	24
Syphilis	9	4	44
Tuberculosis	13	2	15
Liver	6	0	0
Focal Infection	2	0	0
	100	19	

(Analysis of laboratory data omitted.)

ANALYSIS OF THE EYE EXAMINATIONS

As was noted earlier in the paper, the material for this study derives from a series of 100 cases* each of whom showed marked contraction of the form and color fields. This group appeared in the examination of 1900 subjects who comprised all of those patients receiving eye examinations in a consecutive series of 3,000 studied during a period of 5 years.

The technique followed was that required for the use of the Peter Hand Campimeter[8] with a 2° object for form and color fields, a 1° object for blind-spot determination, and the maculae checked with the ½° object for white, blue, red and

*Note. (Together with two more in whom a diagnosis was not established and who, for this reason, were omitted from further discussion.)

green. All cases were rechecked on a Lloyd Slate (small) using a wide-angled hand stereoscope, especially to compare blind-spot findings, as well as determine field contractions within the temporal limits of 20°. The ½° white object was used in the latter procedure, and in most cases larger blind-spots and greater contractions of fields were noted with this smaller test object. Test objects were passed from blind to seeing, and good daylight was the source of illumination.

It is realized that color fields taken by this method cannot be scientifically accurate, so that we have contented ourselves with delineating the red field and noting at the examination where the blue and green limits lay in relation to the former. It is also appreciated that a smaller test object for form, 1° or less, and standard illumination as with the Ferree-Rand perimeter would have given much more accurate data, but so far as the size of the test object is concerned, we have erred on the safe size, namely, that these fields are so much contracted as to come well within our designation. Likewise good daylight gave us rather dependable findings.

TABLE XIX
EYE HISTORY (PATIENTS)

Diagnosis	Glasses	Vision	Inflammation	Photophobia	Miscellaneous
Pituitary	12	11	1	1	7
Thyroid	8	4	0	0	2
Gonad	3	2	0	0	3
Total Endocrine	23	17	1	1	12
Lesion Central Nervous System	6	5	5	2	1
Syphilis	4	1	1	2	0
Tuberculosis	9	0	0	3	0
Liver	1	1	0	0	0
Focal Infection	1	1	0	0	1
Total Non Endocrine	21	8	6	7	2
Total	46	25	7	8	14

Table XIX shows that in the histories of these 100 cases not much eye complaint was obtained. It will be recalled that the ophthalmic examinations were made routinely and not because of eye symptoms. Except for a few cases the need of glasses or some other visual disturbance formed the chief reference to any

eye condition and in practically all of them it was a secondary matter and formed no part of the chief complaint.

In the next table (XX) are certain additional data from the visual record.

TABLE XX

VISION (each eye recorded separately)

	Better Than 20/30	20/30—20/100	Less Than 20/100
Pituitary	51	11	0
Thyroid	17	6	1
Gonad	8	3	1
Total Endocrine	76	20	2
Lesion Central Nervous System	33	5	4
Syphilis	15	3	0
Tuberculosis	18	5	3
Liver	6	4	2
Focal Infection	3	1	0
Total Non Endocrine	75	18	9
Total	151	38	11

Only eleven (11) eyes had really very poor vision. Of these two cases presented retinitis pigmentosa, one in the group of hepatic dysfunctions, the other in that with *tuberculosis*. One case in the group with *central nerve lesions* showed "salt-pepper" pigmentation, with disc changes. A fourth case found in the *thyroid* group had low visual acuity in one eye due doubtless to refractive error.

The next table (XXI) collects the data on pathologic pupils, blind spots, discs and retinæ. As would be expected the greatest pupil abnormalities occurred in the cases classified under lesions of the central nervous system and lues. Enlarged blind-spots were found in the groups in which pressure phenomena or nutritional disturbances were most marked, i. e., twenty-two (22) in pituitary and six (6) central nerve lesions, with eleven (11) in thyroid and seven (7) in the tuberculous groups respectively. Yellow and yellowish[1] discs were found scattered throughout the series, the more yellow being found in cases of retinitis pigmentosa, in liver disease, and with pituitary disorders.

Two cases of elevated nerve heads were found. One, a case of pituitary dysfunction with tumor (B 760), was a little girl of fifteen years with a physical development of ten years but mentally normal. Papilledema of 3 diopters in each eye was found. Later an operation was performed with a fatal result. Autopsy showed congenital cystic tumor of the pituitary.

The other was a young woman of twenty-six, in the group with central nerve lesions having a brain tumor. She showed loss of all of the right field except a reduced mid-nasal quadrant and 10° about fixation and a left field concentrically cut to 20°. Before a final diagnosis of brain tumor was made, the discs were noted to be elevated to 5 diopters and were classified as a questionable papillitis.

Of optic atrophy, only two cases may be so classified. One was a patient of fifty-five with thyroid failure who later suffered a severe head injury with possible fracture. Her fields were seemingly reduced thereby in two years from an initial concentric contraction to 18° to 10° right and about 15° left. The discs were pale but clear cut, of secondary descending type. The contracted fields had so inconvenienced her in getting about that the toes of her shoes were much injured by striking uneven surfaces. The other case was one exhibiting a secondary consecutive atrophy of the left disc, classified in the central nerve lesion group. Her visual field was reduced to a quadrantic type in the lower nasal area about the point of fixation measuring about 15° by 30°, and with vision of 10/40 with glasses.

Vascular lesions were rather unimportant, being mostly of the sclerotic type and found in but a small percentage of the eyes. Likewise exudates played little part in the whole series. Of more importance are the pigmented fundi found in twenty (20)

TABLE XXI

EYE ANALYSIS

Diagnosis	Abnormal Reflexes	Pupils Irregular	Pupils Unequal	Blind Spot Enlarged	Discs Yellow	Discs Yellowish	Discs Atrophied	Discs Elevated	Vascular Lesion	Fundi Pigmented	Exudate
Pituitary	1	0	2	22	6	12	0	2	1	4§	3
Thyroid	1	1	2	11	4	8	2	0	4	0	0
Gonad	1	0	0	2	0	4	0	0	0	0	0
Total Endocrine	3	1	4	35	10	24	2	2	5	4	3
Lesion Central Nervous System	6	2	4	6	4	12	1	2	4	2†	2
Syphilis	3	3	2	4	2	4	0	0	0	8†	0
Tuberculosis	2	0	1	7	4	2	0	0	4	4*	2
Liver	1	0	1	1	4	2	0	0	4	2*	0
Focal Infection	0	0	0	2	2	0	0	0	0	0	0
Total Non Endocrine	12	5	8	20	16	20	1	2	12	16	4
Total	15	6	12	55	26	44	3	4	17	20	7

§ "Snuff" type.
† "Pepper and Salt" Type.
* Retinitis Pigmentosa.

eyes, always bilaterally. Two pituitary cases showed a "snuff" type while one central nerve lesion and four luetic patients exhibited quite characteristic "pepper-salt" pigmentation. Two sisters in the tuberculous group had marked lesions of retinitis pigmentosa as did one in the liver group; these were all bilateral.

TABLE XXII

FIELD CONTRACTIONS

SINGLE EYES

Diagnosis	Cases No.	Concentric Degree 35°—20°	20°—10°	10°	Non Concentric
Pituitary	31	21*	14	26	0
Thyroid	12	10	10	4	0
Gonad	6	2	4	6	0
Total Endocrine	49	33	28	36	0
Lesion of Central Nervous System	21	4*	14	23	4
Syphilis	9	4	4	10	0
Tuberculosis	13	4*	14	7	1
Liver	6	0	8	4	0
Focal Infection	2	2	2	0	0
Total Non Endocrine	51	14	42	44	5
Total	100	47	70	80	5

*One field normal.

Table XXII classifies the contractions. Of nonconcentric contractions, four occurred in lesions of the central nervous system, one of which was the left eye described above, as a secondary consecutive atrophy, another was the right field in the case of possible papillitis of 5 diopters previously mentioned, and one patient with Little's Disease, classified in the central nerve lesion group, exhibited fields below the horizontal midline of about 20°, an altitudinal hemianopic type. The discs in this case showed temporal pallor, and there was marked extraocular imbalance. The fifth irregular field occurred in a tuberculous young woman and was perhaps due to choroidal changes near the disc. This was a left eye, the field being contracted to 20° and not extending below the horizontal midline, also an altitudinal hemianopic type. The right field was concentrically cut to 23°.

Three patients each had one normal field; one in the pituitary group with a left field contracted to 20°, and one in the

tuberculous group had a right field cut to 7°; in neither case could an apparent reason for the monolateral pathology be adduced. The third mono-lateral case was the one referred to previously in the asymetric and secondary consecutive atrophy classification.

A surprisingly large percentage, eighty (80) eyes showed cutting to 10° or less, almost as many from 20° to 10°, while forty-seven (47) were contracted to 35° or less. No cases whose contraction was not at least to 35° were included in this study. The smallest field was found in a married woman of 39 years, in the pituitary dysfunction class whose vision without glasses was 10/15 and Jæger 5 right, and 10/15 and Jæger 8 left, with discs quite yellow, and fields cut to 3° each eye.

Case Protocols—Endocrine Group

In the table below are collected certain of the significant data deriving from the laboratory tests. Reference may be made to these in connection with the consideration of the individual case as discussed below.

TABLE XXIII

Case Number		1 S-1483	2 B-641	3 S-1711	4 B-640	5 S-1735	6 S-1396	7 B-750
Sex		F	M	F	F	F	F	F
Age	Yrs.	35	46	41	10	20	24	30
Height	cm	163.1	182.1	160.4	144.8	163.0	151.9	165.0
Weight	kg	77.0	86.1	100.0	34.1	53.2	69.0	55.8
Weight Deviation	%	+36	+31	+90	—11	+ 0	+30	—14
Lung Volume Deviation	%	—36	+ 1	—30	—15	— 8	—12	—63
Basal Rate Deviation	%	—17	—25	—26	—16	— 9	—39	+47
Blood Pressure	mm	110/66	106/68	112/72	96/48	90/48	90/74	135/70
Pulse Rate	rpm	80	64	64	88	60	51	132
Temperature	0°F	98.4	98.3	98.2	98.0	97.8	97.2	98.0
Alveolar CO2	mm	41	41	44	38	44	38	45
Urine Volume	cc	2030	1030	900	870	710	1310	620
Specific Gravity		1.014	1.025	1.017	1.023	1.019	1.012	1.018
Albumin		0	0	+	+	+	+	+
Casts		0	0	0	0	0	0	0
Sugar		0	0	0	0	0	0	0
Total Nitrogen	gms	12.40	9.68	7.43	6.66	5.80	5.17	5.25
Residual Nitrogen	%	8.1	10.3	13.5	8.7	18.2	9.5	9.3
Phenol Sulphone Phthalein	%	41	59	54	55	42	32	51
Galactose Tolerance								
Normal	gms	40	30	40	20	40	40	40
Observed	gms	60	20	incomplete	10	20	40	5
Deviation	%	+50	—33	normal	—50	—50	+ 0	—87
Blood								
Non Protein Nitrogen	mgm	28	35	29	33	30	32	30
Uric Acid	mgm	3.9	5.3	4.2	4.5	2.4	3.2	4.0
Sugar	mgm	95	82	84	108	99	85	97
Haemoglobin	%	85	80	90	95	90	85	80
Erythrocytes	10^6	4.60	4.20	5.25	5.12	5.72	4.25	4.42
Leucocytes	10^3	10.40	8.70	7.60	6.40	10.00	7.30	8.30
Neutrophiles	%	50	67	73	55	66	49	59
Lymphocytes	%	32	24	24	40	31	40	31
Eosinophiles	%	4	3	3	1	1	2	1
Endothelials	%	0	0	0	4	2	9	8
Miscellaneous	%	0	0	0	0	0	0	1
Wassermann		0	0	0	0	0	0	0
Kahn		0	0	0	0	0	0	0

1. Bilobar Pituitary failure.
2. Pituitary Dysfunction.
3. Pituitary Dysfunction with Syphilis.
4. Pituitary Dysfunction with Tuberculosis.
5. Hypogonad.
6. Thyroid Failure.
7. Hyperthyroid with Henatic Disease.

CASE S-1483. Mrs. K. age 35. Bilobar Pituitary Failure.

The patient's chief complaint was of an increase in the interval between her periods so that the rhythm ranged from two to three months. There was also some metrorrhagia. The patient matured at the age of 14 and at first the periods were irregular. The matter corrected itself, however, and in a short time she established a normal 28-day rhythm with a seven-day period and somewhat scanty flow. Ten months before, however, she skipped a period and from that time on the interval has increased and become irregular.

Family History: Negative.

Marital History: The patient has been married for eight years during which time she had borne three children without miscarriage.

Past History. The patient recorded the usual minor ailments of childhood, and also nephritis in her earlier years. Deliveries were all by Cæsarean section. She developed a breast abscess after her first confinement. She reported a slight loss of aural acuity, some urgency, and occasionally nocturia. The remaining history was substantially normal.

Physical Examination: The patient was a well-developed somewhat obese woman of thirty-five. There were several small telangiectasis on the face, some acne on the shoulders, the teeth were in fair condition only, there was a slight right dorsal scoliosis, minor hypertrophic changes in the finger joints, slight varicosities in the lower extremities, and the routine neurological examination was negative.

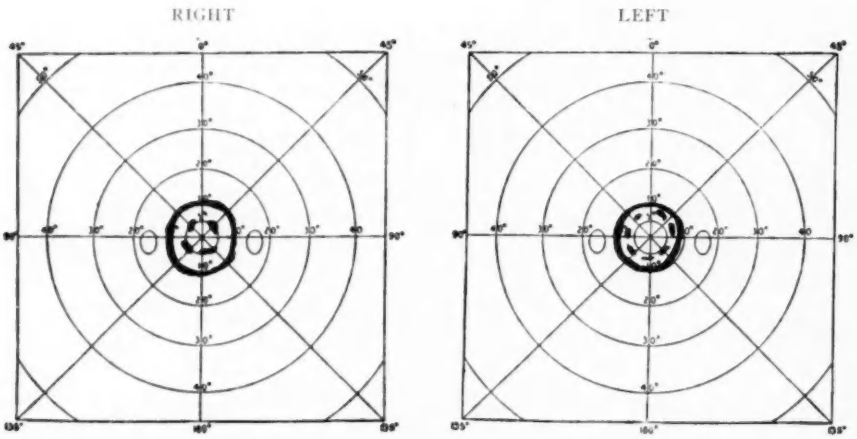
Laboratory Summary: The patient was 36% overweight and exhibited a lung volume definitely below prediction. The basal rate was definitely below the normal, the blood pressure somewhat low, the pulse rate slightly high. The urine volume was ample, the protein intake adequate, the residual nitrogen a high normal. The phthalein test was somewhat low. The sugar tolerance was not completed but as the patient was negative with a 60-gram test meal she was over 50% above her predicted level. The blood uric acid was high and the blood morphology showed a 4% eosinophilia. The serologicals were negative.

Audiogram: There was a slight loss of aural acuity most marked in the speech area.

Radiographic Examination: The sella was normal in shape and size, the right optic foramen was oval in shape and 5x6 mm.

in diameter, the left was circular and 5 mm. in diameter. The sinuses and skull were negative.

Eye Examination: Without glasses vision right =10/15, left 10/10, and Jæger 1 each eye for near point. The pupils showed no pathology and measured 4 mm. The media were clear and the fundi showed only very deep physiologic cupped discs, more marked in the right. Form fields were concentrically contracted to 10° each, color fields slightly smaller.



Concentric contraction of visual fields—Case 1

Opinion: With the patient's obesity tending to a girdle type of distribution, a moderately depressed basal rate with somewhat lowered blood pressure, an ample urine, a normal nitrogen partition, a much increased sugar tolerance, a high blood uric acid, and a 4% eosinophilia, the whole general picture confirms definitely to that of a bilobar pituitary failure.

CASE B-641. Mr. M. age 46. Pituitary Dysfunction.

The patient's chief complaint was of arthritis which was first noticed six months previously, localized in the left great toe. The condition spread involving first the foot, then the left wrist and later the right wrist, ankles, knees, and ultimately practically all of the joints. There was no tenderness but moderate pain on motion especially in the region of the shoulders. For a short time prior to admission the patient had been troubled with vertigo on arising in the morning.

Family History: The father died of arthritis, and the mother of intracranial hemorrhage. Of five brothers three had had rheumatic fever.

Marital History: The patient had been married 21 years but the wife had not conceived during this period. So far as could be ascertained contraception had not been practiced.

Past History: The patient had had the usual minor ailments in childhood, and three years previously a portion of a long uvula had been removed. He was a police officer and in the main had enjoyed excellent health. He complained of an occasional head cold and sore throat but no tonsillitis. He had some edema of the ankles at night. Reported a Neisserian infection twenty-five years previously. He had lost 30 lbs. in the last four months, which fact would indicate an earlier marked obesity. The remaining history was entirely negative.

Physical Examination: The patient was a very well developed, somewhat obese man of forty-six. The hair on the vertex was somewhat sparse, a deviation of the septum with partial obstruction, the tonsils were apparently not offending, there was some tenderness along the cervical spine but no limitation of motion, a slight systolic murmur heard at the apex and transmitted to the axilla was reported, there was some limitation of acute flexion in both knees, tenderness in both ankles, hyperabduction of the arms elicited pain at the inner margin of the head of the humerus, there was some loss of flexion in the fingers of the right hand, the ankles were somewhat edematous.

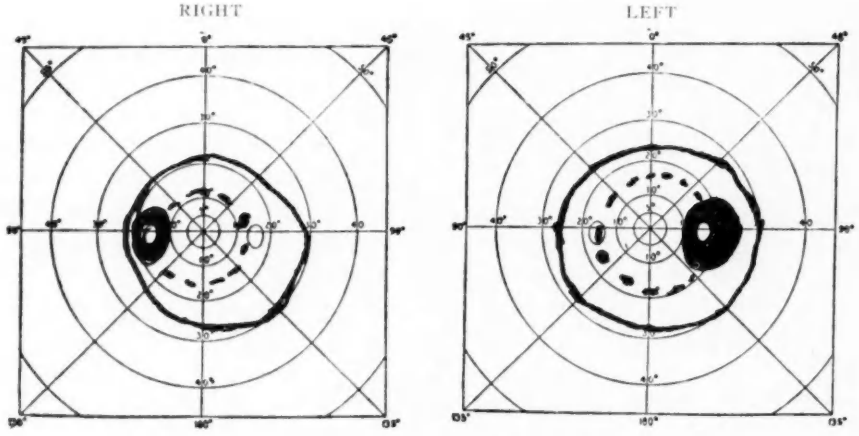
Laboratory Summary: The patient was six feet tall, 31% overweight, his basal rate was 25% below prediction with a somewhat slow pulse and low blood pressure. The urine was substantially normal, the residual nitrogen was high. The galactose was below the normal level. The blood uric acid was significantly above the normal. There was a slight secondary anaemia and a 3% eosinophilia.

Radiographic Examination: The sinuses were negative, there was a deviated nasal septum, the turbinates on the left side were hypertrophied, the sella was normal in size but there was partial bridging of the clinoids, the teeth showed periapical infection and alveolar recession of the lower incisors.

Nose and Throat Examination: The deviated septum was confirmed, the teeth were regarded as doubtful, the tonsils were felt to be nonoffending.

Orthopedic Examination: The findings already reported in the physical examination were confirmed and a diagnosis of infectious arthritis offered.

Eye Examination: Without glasses, vision was 10/8 and Jæger 1 each eye. The pupils were 4 mm., regular and reacted normally. The media were normal, and the fundi showed all vessels somewhat overfilled and somewhat tortuous. Blind spots were considerably enlarged, and the form fields were quite concentrically contracted to 23° and 28° respectively, with colors in proportion.



Concentric contraction of visual fields—Case 2

Opinion: With the patient's physical habits, his lowered basal rate, blood pressure and pulse, the normal urine, increased residual nitrogen, depressed sugar tolerance, high blood uric acid, and 3% eosinophilia, the patient was obviously endocrine and the entire picture is consistent with a pituitary dysfunction of the characteristic formula with the anterior lobe under- and the posterior lobe over-active. There was a definite infectious arthritis, possibly associated with his infected teeth as a complicating factor.

(12 proctols omitted.)

Discussion

It can be well understood that the finding of these contracted fields were of considerable interest to us, particularly because in

only a few cases did the patients register any complaint compatible with the marked narrowing of their visual range. One middle aged woman had been inconvenienced to the extent of having worn out her shoe toes by kicking into unseen steps and other uneven surfaces. One young man had exhibited no difficulty until suddenly entering an elevator which was higher than the floor level. Most of these patients got about surprisingly well considering their handicap. Only those more recently suffering the field loss acknowledged any inconvenience in orientation.

In several, the first tests made, were difficult and misleading because of the shifting tendency which the patient had developed, a facultative defensive efficiency to help overcome their handicap.

The number of cases with frank atrophy of nerve or retina or with a possible element of hysteria (a known complication in one case only, number S-1315 in the protocols) is very small and leaves practically all of the series to be explained upon other bases.

In those cases not presenting some sort of organic interference within the eye itself, the orbit or the cranial cavity, it seems reasonable to assume a casual influence in some nutritional fault either deriving from a local condition or a more general metabolic fault.[12]

The retina is highly organized neuro epithelium somewhat similar to that within the auditory labyrinth.[11] With the known susceptibility of the latter to toxic agents, it seems not impossible that a number of the cases in this series may derive from an analagous mechanism.

Summary and Conclusion

1. In addition to the previously recognized causes of concentric contraction of the visual fields, such as glaucoma, optic and retinal atrophy, hysteria, and an optic neuritis, this study demonstrates that several other disease conditions may produce a like result.

2. Of endocrine conditions disorders of the pituitary not necessarily associated with hyperplasia of the gland would seem to exercise a primary influence. All but a very few of the thyroid and gonad cases showing the eye condition demonstrate a complicating disease condition which is associated with the eye condition. For this reason neither thyroid nor gonad disturbance

is to be regarded as of primary importance in determining the visual phenomenon though they can not be rigorously excluded as factors of minor importance.

3. In addition to the endocrine conditions there is a sufficient incidence of eye changes in lesions affecting the central nervous system, in syphilis, in tuberculosis, and in hepatic dysfunction, to warrant their consideration as probable casual agents of primary significance.

4. The laboratory studies give clear-cut evidence of functional disturbance of the kidney which is broadly to be interpreted as showing an incidence of a disturbed metabolism in excess of similar groups of cases without the characteristic eye findings.

5. From the points noted above it seems reasonable to conclude that in patients demonstrating marked concentric contraction of the visual fields, the possible presence of all of the above should be investigated as well as of the earlier recognized entities in arriving at a diagnosis.

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DISCUSSION

DR. LUTHER C. PETER, Philadelphia: The work from which conclusions are drawn in this communication as a routine measure is to be commended. To make 3,000 studies without a fixed entity for which to search requires an unusual amount of zeal and enthusiasm. However, it is the scientific method of approach which is sure to yield unusual and unexpected results.

Before analyzing these results I feel that the authors will permit me to direct attention to several weak spots in these studies which must be considered in the final analysis. Perimetry at its best is open to some variation, and it is this variation which we are endeavoring to eliminate by standard and accurate methods. Many of the patients included in the report were quite young, and due allowance should be made in the matter of coöperation, in which understanding of exactly what is sought in the examination, in the element of suggestion and its effect upon the results. The latter element is encountered as well in the adult.

In the absence of any marked changes in the fundus, and especially with central visual acuity within normal limits as it was in 76 cases in the series, and in addition, in the absence of marked inconvenience in orientation experienced by some of the patients even when fields were cut to within 10 degrees of the point of fixation, it occurred to me that repeated and most rigid studies should be made to exclude error and psychic influences. These suggestions are not made as a criticism but as a lead to increase the value of this very interesting series of studies.

A further suggestion in an analysis of the material would be the grouping of cases, if possible, with reference to the dominance of a single form of dysfunction or pathology, such as pituitary disturbance, hepatic dysfunction, tuberculosis, etc., to the exclusion of all cases of a complex character. It is manifestly difficult to evaluate the role of each element when several are present, as for example, endocrine disturbance in the presence of nephritic toxaemia. One of the earliest of my studies in ophthalmology was that of the fields in nephritis. The fields are contracted and the blind spots are enlarged to varying degrees in this condition. The same is true in certain forms of endocrine disturbance, with variation peculiar to these forms of toxaemia.

Excluding all errors which might have crept into the technique of the field studies, and the complication of multiple sources of toxaemia and of functional disorder in the same patient, the authors have opened up a field for study which is worthy of much further development. Attention has been called to the effects of toxaemia upon the auditory labyrinth. It is not unreasonable to see an analogy between the toxaemic effect upon the labyrinth and retinal function. The keen observer encounters many cases of retinal and optic nerve dysfunction which disappear when the toxic process is arrested. Toxaemias from the nasal sinuses and the tonsils are frequently observed. Similar visual disturbances can arise from the toxins of tuberculosis, from nephritis, endocrine imbalance and other functional disorders. It is shortsighted to think only of pressure phenomena as one finds it in pituitary disease. One need but recall the number of

cases of sudden visual loss which are mysteriously relieved by simple opening and washing of the sphenoidal sinus without any gross evidence of sphenoidal pathology. It is especially likely that endocrine toxins can bring about similar conditions.

The essayists have directed our attention to a field of study which will be worthy of closest attention in some of our obscure cases of visual disturbance without patent objective cause. A reexamination of some of the cases reported, and further study to eliminate contributing factors, may profitably be a basis for a supplementary report at some further time. They have called attention to the fact that marked field contraction is observed in some conditions other than those usually accepted. The material submitted can most profitably be made the subject of further analysis for more specific and definite data as to the role which each one of the conditions encountered might play in bringing about visual disturbance such as reported. In the absence of histopathologic confirmation, clinical analysis and classification are the more urgent. Further study of even a few of these cases should yield most valuable information as to the exact cause for the marked contraction in each individual case. Such report will crystallize the real value of these comprehensive studies.

Dr. Mark J. Gottleib, New York City: I am not an ophthalmologist, but an otolaryngologist. Last year in my study of progressive deafness I observed that a great number of the cases that were examined had contracted fields, for form and color. I made it a point in talking to a number of ophthalmologists to get their mental attitude toward these field contractions, and the opinions varied. Men to whom I had sent cases and who reported no contractions of the visual fields, on asking them for a chart of the fields they took, I found there were contractions. When I inquired why they were not reported as such they said, "We do not interpret the contraction as significant." Recently Dr. Thomas J. Harris reported in the Annals of Otology that in a large series in which they examined the fields they found no contractions. Drury, in the Laryngoscope for September, found that fields were contracted.

The reason I rise to discuss this paper is that very often we will strike something that is comparatively new, and we ask for a report of the examination, but not an interpretation. We are not able to interpret why these fields are slightly, or moderately, or markedly contracted, but we want this data so that in time, after a large series of cases has been collected, we can more or less correlate the findings.

Apropos of Dr. Rowland's remarks I might say that we are examining the fields at Beth Israel Hospital, New York, of all cases that come into our service, and we are obtaining some very interesting data. For instance, Dr. Rowland reports very interesting field changes and also color changes in the eyes of patients suffering from liver disease. We find that they have contracted fields, some of them, and all of the cases of cholecystitis and cholelithiasis that we have examined have a definite reduction in their hearing.

Dr. William D. Rowland, closing: I want to thank Dr. Peter for his very kind discussion of this paper and his suggestions for further work. In reply to queries raised by the discussors I should like to reply that the eleven (11) children in this series were repeatedly examined, as were all others, until dependable findings were obtained. Interestingly enough, many of the children cooperated better than many adults. Regarding visual inconvenience by contracted fields, it was notable that practically no complaint was elicited from the patient, and a particular point was made of this clinical inquiry. Recently contracted fields always brought complaint from the patient because of orientation inconvenience, but old losses apparently had been adjusted to and no complaint was made about it. Many of these cases have been watched over a period of years and many fields taken. We hope to make further study of selected cases and report our observations.

Dr. Gottleib refers to the work of Dr. Drury in finding contracted fields in cases of deafness. It is a pleasure to state that his work was done in the Evans Memorial Hospital and that the fields were done by us.

Dr. Morton—All the visual fields taken on women were in non-pregnant patients, and always taken between periods. Later there will be reports made on fields taken on pregnant women.

Dr. Howard McI. Morton, Minneapolis: I have but a word to say in this connection. I am very sorry I was too late to hear the address, except the last few words, but I should like to begin my brief remarks by asking Dr. Rowland and his associate if there were observations made as regards the female visual fields in their relation to the state of the uterus, whether gravid, or otherwise, and if records were kept as to how far the state of pregnancy had advanced. Perhaps he spoke of that, but I did not hear it.

Just one point I wish to bring out in this matter. Visual field work of course is still in its infancy, and I am impressed with this—that the visual field does not simply express a light sense, or a color sense perception, but that we have a series of perceptions all correlated and intimately associated, and mutually interdependent. We cannot take a visual field without having one or the other of these related factors present. When we begin to increase the field intensity we introduce a chronic element that in itself varies somewhat as the intensity varies.

As the authors have said, these cases are only suggestive, something to give us food for thought and some inspiration for activity. But something more definite might be obtained. I have done considerable work along this line, and am now examining the visual fields, at a given point, under different light intensities. For instance, through these investigations we should accurately establish, under a given illumination, a normal, and then find out in these different groups, under two or three different intensities of illumination, how the tests vary. In this way, what might at first appear as a group of similar cases might be found to be in fact dissimilar. I think we must take into account more and more the importance of the light sense in its correlation with the form sense.

THE FREQUENCY OF SYMPATHETIC OPHTHALMIA

GEORGIANA DVORAK-THEOBALD, M. D.

CHICAGO, ILL.

FROM THE PATHOLOGICAL DEPARTMENT OF THE ILLINOIS EYE AND EAR INFIRMARY

The cases here reported were selected from the records of the Illinois Eye and Ear Infirmary, covering a period of twenty years. The clinical diagnoses were made by the staff surgeons.

During this period of twenty years, 216,592 patients presented themselves in the eye department for treatment. Of these 24,404 were in-patients and 192,188 were out-patients, and 1,465 had a history of perforating injury. The following table, (Table I) shows the distribution of the injuries according to age and sex:

TABLE I. AGE AT WHICH PERFORATION OCCURRED

Age	0-10	11-20	21-30	31-40	41-60	61-85	Total
'09 to '14	M.69 F.29	M.79 F.12	M.115 F. 7	M.92 F. 2	M.125 F. 4	M.18 F. 1	498 55
'14 to '19	M.61 F. 22	M.66 F.10	M. 43 F. 8	M.39 F. 3	M. 56 F. 4	M.21 F. 4	286 51
'19 to '24	M.57 F.27	M.56 F.11	M. 60 F. 6	M.48 F. 3	M. 71 F. 6	M.18 F. 2	310 55
'24 to '29	M.45 F.11	M.39 F.11	M. 22 F. 3	M.14 F. 4	M. 31 F. 12	M.16 F. 2	167 43
Total	321	284	264	205	309	82	1465
Occurrence of Sympathetic Ophthalmia	4	1	1	1	2	1	

It is interesting to note that since the Workman's Compensation law was passed in 1917 the number of perforating injuries in male patients between the ages of 21-60 decreased markedly. The Infirmary is a State charitable institution and refuses admission to patients who come under this law.

492 eyes were enucleated with a lapse of time following injury, ranging from 48 hours to 40 years. (See Table II.)

TABLE II. LAPSE OF TIME BETWEEN INJURY AND ENUCLEATION.

Within 48 Hours	Within 4 Weeks	Within 4-8 Weeks	Within 2-12 Months	After 1 Year to 40
6	78	87	123	198

During these 20 years 7,444 cataract extractions were performed and 2,922 other intra-ocular operations, including iridectomy and trephine. There were twenty-two enucleations after cataract extraction and twenty-nine enucleations after operations for relief of intra-ocular tension.

Thirty-one diagnoses of sympathetic ophthalmia were made. Of this number, twenty-three primary eyes were enucleated. The remaining eight refused consent to operation or wandered elsewhere. These patients showed symptoms in the second eye, the most important of which were ciliary injection, precipitates on the posterior surface of the cornea, cloudy iris pattern and posterior synechiæ.

Forty-eight eyes were enucleated with the diagnosis of sympathetic irritation. These patients had symptoms in the second eye of lacrimation, pain, slight circumcorneal injection and blepharospasm. Many histories mentioned dimunition of vision, but this was probably due to lacrimation and blepharospasm.

Of the twenty-three cases enucleated with a clinical diagnosis of sympathetic ophthalmia, eleven eyes proved histologically to be so. Two eyes were chronic endophthalmitis with a mild infiltration of plasma cells. Nine were atrophy following endophthalmitis, and two post-traumatic chronic infiltrating iritis. (Iritis Serosa, Fuchs.)

Among the forty-eight cases of sympathetic irritation were eleven endophthalmitis with beginning atrophy, fourteen cases of traumatic iridocyclitis, and twenty-three atrophy bulbi which show fresh lymphocytic infiltrations. These infiltrations are probably responsible for the irritation in both eyes, as Zimmerman has pointed out. (Zeitschrift for Augenheilkunde, Vol. 57.) The iridocyclitis cases showed varying degrees of round cell infiltration in the uvea and retina, with some fibrinous exudate.

I shall not give a detailed history of the cases of sympathetic ophthalmia, but shall tabulate them in the order in which they were received in the laboratory. In constructing this chart, I followed the headings used by Professor E. Fuchs (A. f. O., lxi, 1905), adding the laboratory number, occupation and service on which treated. (See Table III.)

TABLE III.

Lab. Number	Name	Sex	Age	Occupation	Service	Cause of Sympathetic Inflammation	Lapse of time between injury and beginning of symptoms	Time between beginning of symptoms and enucleation	Course of Inflammation and ultimate vision.
1605	Geisler	M.	7	School	Nance	Struck with unknown object ½ pearl button found in eye	5 weeks	5 weeks	Severe 10/200 V.
1621	Heekin	F.	8	School	Wilder	Rifle discharged Multiple Perforation	4 weeks	3 days	Severe 20/70 V.
1858	Worsham	M.	18	Clerk	Dodd	Perf. injury Linear extract. Blow by fist	2 years after first injury	11 days	Severe 20/200 V.
1878	Sward	M.	34	Laborer	Lane	Perforation by chip of iron from hammer	3 years	8 days	Severe 3/200 V.
1902	Binder	M.	8	School	Findlay	Paring knife penetrated limbus	5 weeks	2 days	Mild 20/20 V.
2024	Cissna	M.	8	School	Barr	Rupture by blow	2 months	?	Very severe fingers at 1 ft.
2131	Molitor	M.	52	Blacksmith	Woodruff	Cataract ext. followed by inf.	4 weeks	6 days	Mild 20/200 same as before op.
2440	Card Missing	?	?	?	?	Central cornea perforation	?	?	?
2427	Gustavson	M.	56	Laborer	Goldenburg	Cataract ext. attempted	8 weeks	19 days	Very severe V. not recorded
2566	McNamara	M.	62	Fireman	Lebensohn	Trephine iridectomy	15 weeks	4 days	Mild 20/20 V.
2594	Ray	M.	24	Miner	Orcutt	Rupture of sclera by pool cue	4½ weeks	11 days	Mild w gl. 20/15+3

The sympathetic inflammation followed perforating injuries in eight cases, cataract in two and iridectomy for glaucoma in one.

The following table shows the percentages:

TABLE IV.

	Perforating Injuries	Cataract Ext.	Other Intra-ocular Oper.
No. of cases	1465	7444	2922
No. of Enucleations	492	22	29
Cases of Sympathetic Ophthalmia	8	2	1
Frequency	0.54%	0.03%	0.029%

The following table shows the percentage of sympathetic ophthalmia found by various authors: this table was compiled from Peters (Handbuch) Gradle (American Encyclopedia of Ophthalmia) and Parsons (Pathology). There is no uniformity in the manner in which these percentages were obtained so comparison would be of little value. (Table V).

Referring back to Table I, it will be seen that four of these cases occurred before the age of ten years and the others were evenly distributed over the following decades. It is the opinion of some authors that sympathetic ophthalmia is now less frequent. Our statistics do not show this as the number of perforating injuries is less in the second ten years, corresponding to the decreased number of sympathetic cases.

The following chart shows the distribution of the cases over twenty years.

TABLE VI.

'09	'10	'11	'12	'13	'14	'15	'16	'17	'18	'19	'20	'21	'22	'23	'24	'25	'26	'27	'28
0	0	0	2	0	1	2	1	0	1	0	0	0	0	2	0	1	1	0	0

I need not give the full details of the histological picture of these cases as it has been done many times since Shirmer's monograph on Sympathetic Ophthalmia in 1899 (Shirmer, Graefe

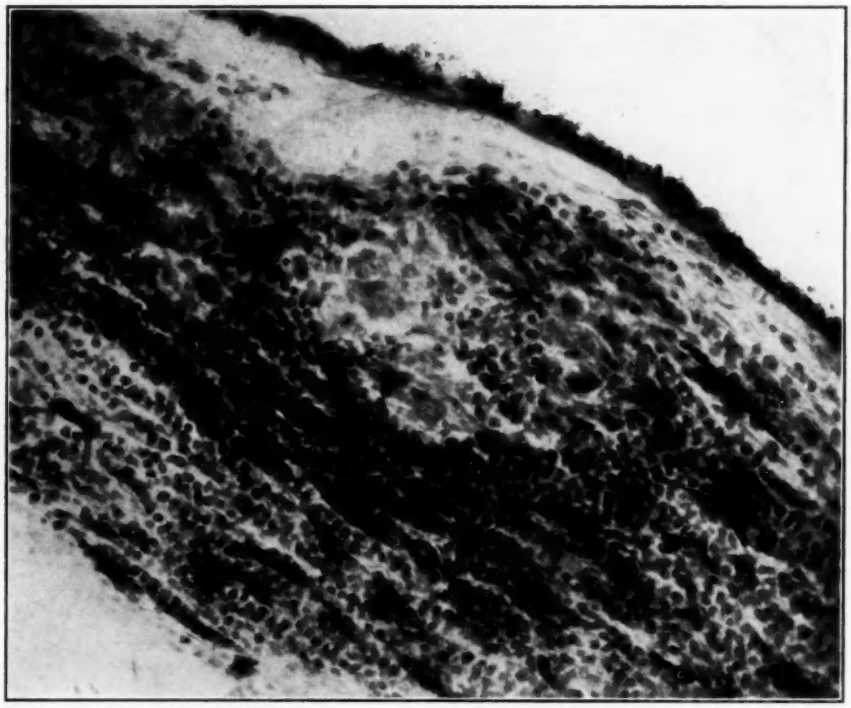
Saemisch Handbuch d. ges. Augenhelk., second edition) and Professor E. Fuchs' paper in 1905. But I would like to point out some important facts on differential diagnosis between sympathetic ophthalmia and post-traumatic chronic infiltrative inflammation and present to you a few interesting details I found in my slides.

TABLE V. PER CENT OF SYMPATHETIC OPHTHALMIA FOUND BY DIFFERENT AUTHORS

Author	Per cent of Sympathetic Oph.	How Obtained
Mooren	0.134	% of all clinic patients
Becker	0.15	% of in-patients
Collins	0.9	% of in-patients
Cohen	0.135	79 in 58484 patients
Steindorf	0.33	42 in 1255; 30 after sev. injury
Ohlemann	2.0	% of infected perf. wounds
Knies	3.0	
Hobby	3.5	% in perf. wounds
Szily, Arch. j. Augen., 13:34	0.00	in 1092 injuries
Szily, Arch. f. Augen., 13, 1. S. 33	0.6	1 in 150 injuries
Kitamura	2.94	17 in 577 perf. injuries
Weigelin	1.00	12 in 1150 perf. injuries
Doman	3.10	387 perf. inj., 103 in children
Reis	1.2	6 in 500 perf. injuries
Krebs	6.	11 in 181 intraocular f.b.
Fuchs	13.	24 in 180 injuries
Von Bull		8 in 17 magnet extraction
Von Paly	10.	86 in 45 diseases of uv. tract
Author	0.54	8 in 1465 perf. injuries
	0.03	2 in 744 cat. extr.
	0.029	1 in 2922 other intraoc. oper.

2566 is a classical picture of infiltrative and proliferative uveitis. Fifteen weeks after a combined trephine and iridectomy the patient returned to the hospital complaining of diminished vision in the second eye, and showing a fine ciliary injection accompanied by many brown deposits on the posterior surface of the cornea. The sections show a uniform infiltration throughout the uveal tract. It has the characteristic arrangement of the in-

filtrating cells of sympathetic inflammation. In the iris the outer dark staining zone is formed by lymphocytes, and between it and the pigment epithelium is the light staining area formed by epitheloid cells. In the ciliary body the unpigmented epithelium is separated from the pigmented layer by the same infiltration. It is well preserved, while the pigmented layer is markedly destroyed. In the choroid the characteristic infiltration occupies the outer layers, while the chorio-capillaris, lamia vitrea and pigment epithelium are well preserved.



2566—Typical granulation tissue of Sympathetic Ophthalmia in choroid.

To show the clinical similarity and the anatomical difference between sympathetic ophthalmia and post-traumatic infiltrative uveitis, I would like to cite a case from the service of Dr. Harry Woodruff, Laboratory No. p. 618.

Three or four weeks following an iridectomy for glaucoma, the second eye became injected and showed the usual symptoms of sympathetic ophthalmia. The injection was slight, lacrimation

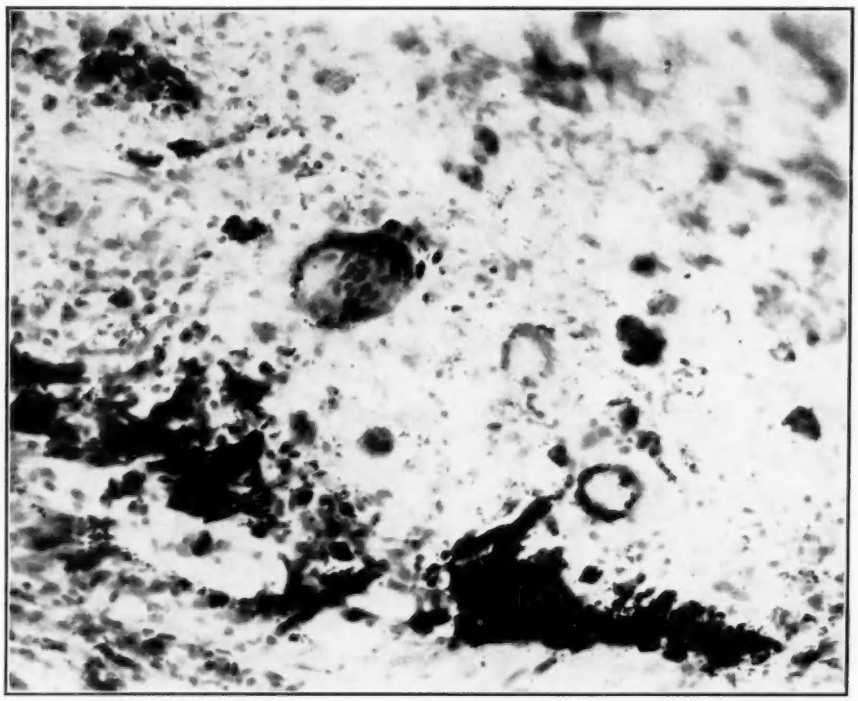
not noted. Photophobia and diminished vision were complained of. Deposits were present on the posterior surface of the cornea, the iris markings were blurred and many posterior synechiæ developed. Treatment consisted of injections of autohemic serum which apparently arrested the progress but did not improve the condition. The primary eye was removed eight months after the iridectomy. Serial sections were made and carefully studied for characteristic granulation tissue, but none was found. The following is a description through the coloboma area:

2566—Ciliary body, showing typical granulation tissue of Sympathetic Ophthalmia.

The scar of operation is just posterior to Schlemm's canal. There are many superficial and deep vessels throughout the cornea. The anterior chamber is very shallow, and is wanting in places where the iris is tumified and touches the posterior surface of the cornea. The pupillary end of the iris is fixed to a pupillary membrane which is in close apposition to the lens capsule. This membrane reaches to the scar of operation and to the

ciliary processes, pulling and stretching them forward. The ciliary portion of the iris is markedly tumified and shows a dense infiltration of round cells, especially in the posterior layers. These cells are lymphocytes and plasma cells. No epitheloid cells were found. The pigment layer of the iris shows destructive changes; it is partly or entirely wanting, partly proliferated. The pigment epithelial cells have migrated through the stroma of the iris and in many places reach the surface of the anterior limiting membrane. The posterior chamber is filled with connective tissue, a continuation of the thick pupillary membrane. This membrane continues back to the ora serata. This membrane, too, is infiltrated with round cells. The infiltration continues back into the ciliary body, more marked in the processes. The pigment epithelium shows the same changes as in the iris. The same cellular infiltration is found hugging the ciliary processes and destroying the unpigmented epithelium. In parts the unpigmented epithelium is entirely destroyed. The infiltration continues into the choroid, with the same cells and the same degeneration and proliferation of pigment cells. The lamina vitrea is missing in many places. In the sub-retinal space are found occasional heaps of cells with more or less pigment content. Pigment cells are found migrating into the retina. The outer layers of the retina show changes of degeneration, the rods and cones being disintegrated especially opposite the site of the more dense choroidal infiltration. Upon the nerve fibre layer there is a layer of connective tissue, increasing in thickness and covering over the optic disc. The optic nerve shows changes of secondary optic atrophy.

In the following case, 2594, it was difficult to reach a conclusion as to diagnosis. A few sections studied led to the diagnosis of post-traumatic chronic infiltrating uveitis. Later serial sections were studied and a single focus of proliferating epitheloid cells with a few giant cells was found in the ciliary body. Four and one-half weeks after the right eye was ruptured by a blow with a pool cue, the left eye showed a faint ciliary injection and small deposits on the posterior surface of the cornea. Vision was then 20/30. In spite of treatment the deposits increased and vision diminished. Eleven days after the symptoms began the right eye was enucleated.



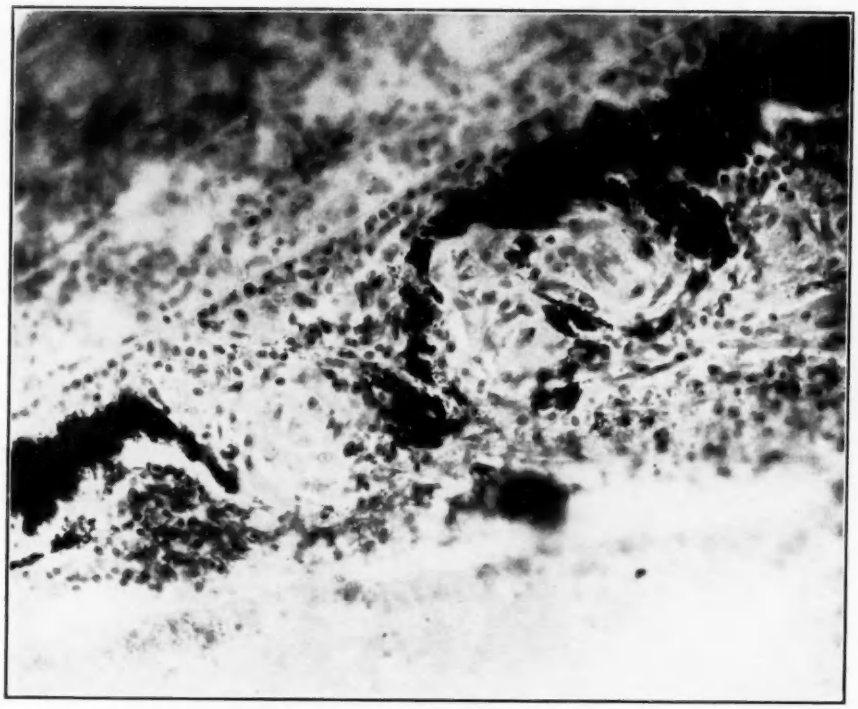
2594—Ciliary body—Focus of epitheloid and giant cells.

The sections, 2594, show the iris was torn from its attachment and doubled up on itself in the scleral wound which is just back of the limbus. The lens is absent. The iris is atropic and its pigment epithelium is degenerated. It is slightly infiltrated with round cells, and round cells infiltrate the connective tissue that surround it. A thick band of connective tissue extends from the healed wound to the ciliary processes on the opposite side pulling them forward. The anterior chamber is partially filled with blood. The ciliary body is atrophied and contains foci of round cells. In places its pigment epithelium is migrated. In the ciliary body on the side of the wound was found a single focus of epitheloid cells with a few giant cells. This would have been missed if serial sections had not been studied. On the side opposite the wound is a hemorrhage in and behind the choroid. This is probably the site of the blow. The choroid is uniformly infiltrated with round cells, which are lymphocytes and plasma cells. There is a small hemorrhage in the anterior vitreous undergoing absorption as evidenced by the many foreign-body giant

2594—Round cell infiltration in choroid.

cells bearing blood pigment, found in its vicinity and throughout the vitreous. Plasma cells are found scattered in the vitreous and lying on the retina. There is a slight infiltration around a few retinal vessels.

Case 1902 presents the most interesting detail of an early stage of sympathetic ophthalmia, which was first described by Dalén and later interpreted by Professor E. Fuchs. The latter found this condition in four of his cases and emphasized its importance in the early diagnosis of sympathetic ophthalmia. These characteristic Dalén-Fuchs areas consist of small proliferations of the cells of the pigment epithelium of the uveal tract. In our case they are found in the ciliary body, symmetrically located. The layer of pigmented cells is separated from the lamina vitrea by heaps of cells with much protoplasm, large oblong nuclei and one or two distinct nucleoli. These epitheloid cells are derived from the pigment epithelium. This proliferation extends beyond the pigment layer, separating it from the unpigmented layer.



1902—Dalen-Fuch's areas. Epitheloid cells replacing pigment epithelium of ciliary body.

Drawings of Dalen-Fuch's areas, from Fuch's (v. Græfe's Archer. Bd. LXI.)

The clinical diagnosis of sympathetic ophthalmia presents many difficulties. Parsons (Pathology of the Eye, Vol. 4, 1908) says that "the appearance of precipitates on the back of the cornea of the second eye is proof that sympathetic ophthalmia has already supervened." Adelbert Fuchs (Atlas of Histiopathology of the Eye) tells us that "chronic infiltrating post-traumatic iritis is characterized clinically above all by precipitates, resembling in this way sympathetic ophthalmia." It is possible that in the cases where the primary eye was an atrophy bulbi, the inflammation in the second eye was due to a focal or systemic infection, and had no connection with the injury.

A. Fuchs makes a nice anatomical differentiation between three clinically similar conditions. First, endophthalmitis effects the tissues lining the vitreous cavity, namely, the retina, ciliary body and iris. It is an exudative inflammation, the exudate being composed of polynuclear leucocytes. When the endophthalmitis is of long standing plasma cells are found in the tissues and also in the exudates. Second, in post-traumatic infiltrating chronic iritis the same tissues are affected, but these tissues are infiltrated with mono-nuclear cells. All of our cases had more or less cellular infiltration in the choroid. Third, sympathetic ophthalmia is an infiltration and proliferation within the uvea itself.

Shirmer's (G. S. VI 2, 1900) statement that the chief difficulty in dealing with sympathetic ophthalmia is that it is always a presumptive diagnosis, still holds true.

Summary

1. Our small statistics show the greatest frequency of sympathetic cases occurring in the first decade. There are four cases below the age of ten, and only one case for each of the following decades.

2. These statistics show that sympathetic ophthalmia is actually much less frequent than the enucleations which are done for sympathetic or for fear of sympathetic ophthalmia. However, the enucleations are justified, for, with our present clinical knowledge, we have no certain means by which to decide, in an early stage, whether it is sympathetic ophthalmia or a less harmful infiltrating inflammation.

3. Diagnosis can be made only histologically. This may present some difficulty as case 2594 shows. For this purpose each eye must be sectioned serially and at least every tenth section carefully examined.

DISCUSSION

Dr. Walter E. Camp, Minneapolis: In this splendid paper Dr. Theobald has brought several important facts forcibly to our attention. First, the importance of keeping good case records and histories, not only in private practice but also in clinical work; second, the importance of obtaining, whenever possible, pathological specimens and sectioning them for diagnosis and study.

Statistics show that the incidence of perforating injuries of the eyeball has decreased during the past ten years in all age groups. This decrease in perforating injuries is accompanied by a correspondingly greater decrease in the incidence of sympathetic ophthalmia. This percentage decrease in sympathetic ophthalmia is to be accounted for by more prompt and efficient treatment of traumatic cases during the past decade. The development of more rigid aseptic technique; improved methods of foreign body extraction; the introduction of foreign protein therapy and possibly more radical surgical procedures have all been a factor in reducing the incidence of sympathetic ophthalmia.

At Moorfield Hospital from 1884 to 1898, a period of fourteen years, every 151st case admitted was sympathetic ophthalmia. In a period of twenty years from 1909 to 1929 in Dr. Theobald's series, every 787th case admitted as in-patient was diagnosed clinically as sympathetic ophthalmia. In the older ophthalmic literature the incidence of sympathetic ophthalmia in cases of perforating wounds varied from 1 to 2 per cent. In Dr. Theobald's series it is approximately one-half of 1 per cent.

Dr. Theobald has clearly brought out the difficulties in making a diagnosis of sympathetic ophthalmia clinically. Only 50 per cent of the cases diagnosed clinically as sympathetic ophthalmia proved so on pathological examination. The remaining 50 per cent were acute exacerbations of an old endolphthalmitis or uveitis serosa characterized by lymphocytic infiltration causing irritation in the good eye. There is no one symptom or group of symptoms clinically that is pathognomonic of sympathetic ophthalmia and when there is any doubt, enucleation of the exciting eye should be advised.

As Dr. Theobald has explained, not only the clinical diagnosis, but also the pathological diagnosis may be quite difficult, especially if serial sections are not made. In advanced cases of sympathetic ophthalmia the whole uveal tract is densely infiltrated with round cells, epitheloid cells and giant cells. In the earlier cases the characteristic pathological feature is a nodular epitheloid cell deposit involving the posterior surface of the cornea, the iris, ciliary body and choroid. The pathology in the sympathizing eye is identical with that of the exciting eye.

By far the greater percentage of cases of sympathetic ophthalmia are found in children during the first decade, because of the greater number of perforating injuries during that period. Four of Dr. Theobald's series of eleven cases were found in children under ten years of age. Parsons believes that aside from the great frequency of injuries in children, they are naturally predisposed to sympathetic inflammation.

The most favorable conditions for the development of sympathetic ophthalmia, or, in other words, the history and clinical findings in a typical

case, would be as follows: Child, age 6, perforating wound of globe in region of ciliary body with prolapse of some portion of the uveal tract; severe plastic type of reaction; little or no suppurative or exudative type of inflammation. From six to twelve weeks later, ciliary injection develops in the sympathizing eye, fatigue or loss of power of accommodation, posterior deposits on the cornea, severe plastic uveitis, and markedly reduced vision. Treatment is of little avail except in checking the advance of the disease.

Many variations occur from the above typical case, and one must always be alert and try to differentiate clinically, if possible, between sympathetic inflammation and coincident inflammation or irritation of a benign nature in the good eye.

In closing, I wish to congratulate Dr. Theobald on this excellent piece of work.

DR. ALLEN GREENWOOD, Boston: Your President has given me permission to call your attention to a little sentence which was spoken this morning by our guest of honor. Some of you may not have been here at the time, or some may not have appreciated what was said. To me it was the finest thing that has come under my observation for some time and shows that our honor guest is one of the many humanitarian physicians practicing in this world. The sentence is this: "I frequently have my patients come back to give them the *solace of an optimistic report* in regard to their condition."

I wish to congratulate Dr. Theobald on her work and also to call attention of the members to the merit of this new member of the gentler sex coming before you, and also to impress upon you the wonderful chance a woman has in the type of work that Dr. Theobald is doing.

In regard to the paper itself: During the World War I received from every ophthalmic surgeon in France a record of every patient under his observation every month. Only one case of sympathetic ophthalmia was ever included in these reports, a case that came under the observation of Dr. Ralph Fenton. Why, with the thousands of penetrating wounds of the ciliary body during the war, did we have but one case of sympathetic ophthalmia? Some thought it might be due to the fact that every soldier received a good dose of anti-tetanic serum. Another thing of course, the badly injured eyes were removed quickly. I have long had the feeling that a case of true sympathetic ophthalmia is exceedingly rare.

I want to call attention to one thing. I received a letter from an ophthalmologist in a distant county seat asking me when an injured eye should be removed to prevent sympathetic ophthalmia. I sent back this message—that if the injured eye was absolutely free from all signs of iridocyclitis or irritation he need not worry. I think that was good advice to give him.

DR. GEORGIANA DVORAK THEOBALD, closing: I wish to thank Dr. Camp and Dr. Greenwood for their very kind discussion of my paper. The belief that sympathetic ophthalmia occurs much more frequently than is shown by my figures dates back to the report by German authors follow-

ing the Franco-Prussian War in 1870 that all such cases following injury were sympathetic. The reports were accepted without being confirmed by study of the subject. However, the late War led to a re-study of the early work and abandonment of the early position. The fact that that abandonment has not been sufficiently broadcast is one justification for this paper. The frequency of real sympathetic ophthalmia further brings up the question of the alleged cure by serum and other means. The diagnosis can only be confirmed by the aid of the microscope, and until there is definite proof, any treatment with serum or other means must be accepted with hesitancy. I believe the use of vaccines and serums at the time of injury aids much in reducing the number of cases of sympathetic ophthalmia.

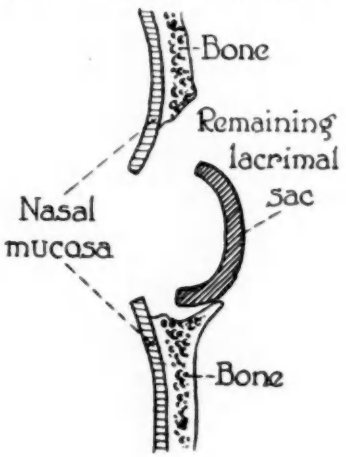
CONSERVATION OF THE LACRYMAL SAC

F. N. KNAPP, M.S. Opth., M.D.

DULUTH, MINN.

The removal of the lacrymal sac has been practiced continuously since re-introduced by Berlin in 1868. This operation relieves empyema of the sac. Its removal is always followed by lacrymation at least during cold weather.

In 1904, Toti devised a combination of extirpation and window resection of the lacrymal fossa to the nasal cavity with destruction of most of the mucous membrane of the sac. (Fig. 1). This procedure was to re-establish the flow of tears through



Toti's operation

Fig. 1

an artificial canal. Toti's dacryocystorrhinostomy was the beginning of an era of conservation of lacrymal sac drainage. His technique has been modified by Kuhnt and others. In 1911, West described his intranasal operation of dacryocystorrhinostomy. This is a more extensive removal of the lacrymal sac than Toti's procedure. Mosher, Wiener and Sauer, and many others,

have introduced modifications of West's intranasal operation, All of these operations, whether endonasal or external, have partially resected the lacrymal sac and left a wound through the nasal mucous membrane that heals by the formation of scar tissue. The opening must be treated by drains, probes, stylets or by radium as recommended by Withers. Mosher says, "All intranasal methods must face the danger of reclosure of the canal, and that every operation on the lacrymal and nasal duct is only a temporary success, unless supplemented with frequent probing or the prolonged wearing of a stylet." Operations that enter the sac through an incision of the canaliculus will fail as the function of lacrymal drainage is thereby destroyed.

The technique of Halle, Benedict, Wiener and Sauer do not destroy a large part of the sac thereby preventing the closure of the cavity by granulation tissue.

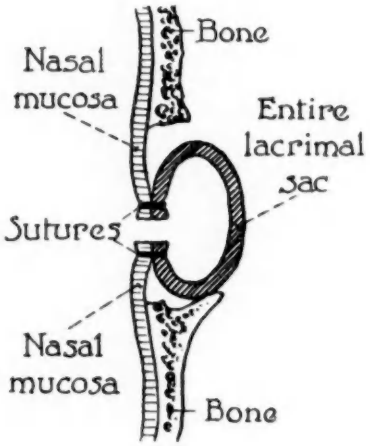
The technique of Ciricione utilizes all of the lacrymal sac. Burch's procedure is along similar lines. Postoperative treatment necessary even in this method to continue the opening into the nasal cavity without scar tissue formation.

Any procedure that slits or destroys the canaliculus, as advocated by Pratt, Green or Prince, will defeat its own purpose. No lacrymal drainage is normal after this procedure.

Direct anastomosis of the lacrymal sac to the nasal mucous membrane by sutures permits the wound to heal by first intention. In plastic dacryocystorrhinostomy of Dupuy-Dutemps and Bourguet none of the nasal mucosa nor lacrymal sac are destroyed. Only the bone separating the two mucous layers is removed. (Fig. 2). Tjanides prefers the term ascorhinostomy, meaning a union of the lacrymal sac to the nasal cavity.

Many cases of epiphoria are due to other causes than obstruction of the lacrymal canal. Capillary drainage is lost by eversion of the lids after injury or chronic thickening of the lid margins. Obstruction of the canaliculæ prevent the re-establishing of drainage in the lower part of the lacrymal system.

Tuberculosis of the lacrymal sac or new growths are contraindications for dacryocystorrhinostomy. Trachoma is a contraindications for surgical interference of the lacrymal sac. Tumors of the sac should be treated by radical surgery or by radium and roentgen rays. Tuberculosis or tumors of the lacrymal sac are comparatively rare.



Dupuy-Dutemps & Bouget

Fig. 2

Ulcer serpens may require a hasty removal of the lacrymal sac to remove the foci of infection.

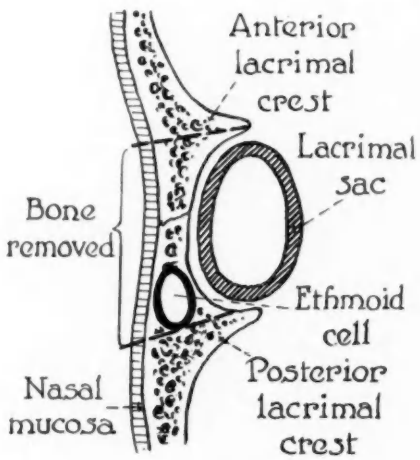
All operative procedures within the globe must be antidated by the elimination of the danger of infection from the lacrymal sac. Extirpation of the sac either by the external route or by this endonasal route will eliminate the danger of infection from the lacrymal sac. Extirpation of the sac results in epiphoria. When time permits, the infection may be removed and epiphoria eliminated at the same time by plastc dacryocystorrhinostomy.

Many cases of epiphoria may be cured by conservative treatment as described by McCoy. The most effective simple measure is dilation of the upper and lower punctæ which is often the result of chronic thickening of the lid margins. This produces a slight ectropion with loss of capillary suction. After dilation the sac may be filled by a solution of holocaine and adrenalin to reduce the congestion in the lacrymal sac and canal. Many cases are cured by this simple treatment. Recent cases of obstruction of the lacrymal canal may be cured by the passage of Bowman's probes without slitting the lower canaloculi. Probing is often a temporary cure, and the majority of these cases will suffer later from obstruction. The use of albolene has been advocated. It

has its dangers. Recently sea tangle has been described by Brown as an effective measure for the relief of lacrymal obstruction.

After entering the nasal cavity from the lacrymal fossa, one soon learns of the extreme variations in the position of the middle turbinate as well as the location of the anterior ethmoid cells. Onodi has demonstrated cells lying in the immediate vicinity of the lacrymal fossa. (Figure 3). A cell will be found directly under the lacrymal fossa in about one-half of the cases. The frontal sinus may even come down below the lacrymal fossa. An enlarged middle turbinate may overhang the lacrymal fossa and prevent drainage. In such a case the tip of the *turbinate* should be removed. A previous incision of the canaliculæ will lessen the normal flow of tears following operation. This procedure is very rarely warranted and in most cases destroys the function of the entire lacrymal apparatus in that it eliminates the contact of the punctum to the bulba conjunctiva. In chronic dacryocystitis, the draing of pus and the restoration of the flow of tears will be relieved by anastomosis of the tear sac to the nasal mucous membrane.

It is necessary to establish a large opening to the nasal cavity. If this opening is a little larger than the size of the com-



Ethmoid cell
(modified from Onodi)

Fig. 3

bined puncta of the upper and lower lid, drainage will be established.

Fluoroscopy of the lacrymal sac after the injection of lipiodol will readily demonstrate total obstruction of the lacrymal canal. At the same time the size of the sac is determined.

Plastic dacryocytorrhinostomy is indicated for the relief of chronic lacrymation caused by the obliteration of the lacrymal canal with or without pus; for the cure of a mucocele resulting from sinus disease, trauma, or syphilis with malformation of the bony lacrymal canal.

According to O'Brien, "Procaine hydrochloride, in a strength of 2 per cent in physiologic sodium chloride solution, is practically a perfect anesthetic for infiltration anesthesia and nerve block. It should be freshly prepared, sterilized in the autoclave or by boiling, and combined with epinephrine just prior to injection. Before injection, one should decide on the amount of solution to be used and add epinephrine in quantities such that the total amount received by the patient will not be more than 8 to 10 minims (0.5 or 0.6 cc.)"

Into the sac is instilled a few drops of this solution by a lacrymal syringe, through the lower canaliculus. The skin along the line of incision is infiltrated. A deep injection is made above the internal palpebral ligament to block the supratrochlear and infratrochlear nerves. Another injection below blocks the infraorbital nerve. The nasal mucous membrane is anesthized opposite the lacrymal fossa by placing a tampon of equal parts of a solution of cocaine hydrochloride 10 per cent and adrenalin chloride in front of the middle turbinate.

The skin incision starts at the level of the insertion of the internal palpebral ligament and 3 mm. mesial to the ligament. (Figure 4). The first half of the skin incision must be vertical. The lower half of the incision is curved parallel to and three millimeters below the infraorbital margin. This direction should be followed carefully, to correspond to the folds in the skin and prevent a noticeable scar. The dissection through the superficial fasca, orbicularis palpebrarum muscles and deep fasca is the same as for extirpation of the sac. Horner's Muscle which is the posterior half of the internal palpebral ligament is left intact. The muscle fibres are separated, but not cut.

Incision thru skin

Fig. 4

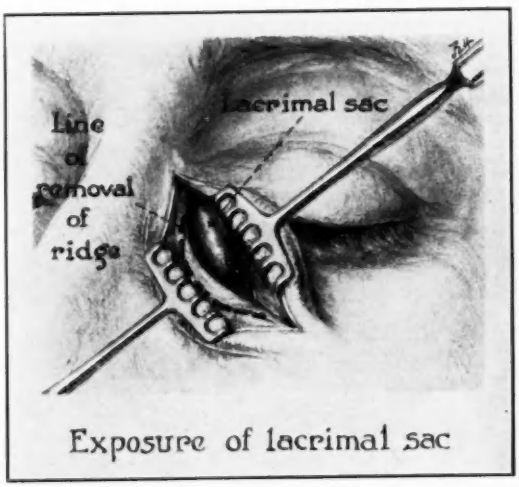

Lacrimal sac
Line of removal of ridge
Exposure of lacrimal sac

Fig. 5

The mesial half of the sac is separated from its fossa and held toward the globe by a flat retractor. If the lacrymal sac is extremely thin the separation may include the periosteum lining the lacrymal fossa. This periosteal dissection should be carried downward to the upper limit of the lacrymal canal for three to five millimeters. The periosteum is separated externally from the bone exposing the lacrymal crest. The lacrymal crest is completely removed by a chisel. (Fig. 5). This allows easier entry to the lacrymal fossa. A small opening through the bony fossa is first made with a chisel, exposing the nasal mucosa which is at once elevated in all directions. The entire bony wall of the lacrymal fossa is removed from the anterior to the posterior crest, and from the internal palpebral ligament above to the beginning of the lacrymal canal below. This is best done with a Citelli forceps.

In the original operation, as described by Dupuy-Dutemps and Bourguet, the incision of the lacrymal sac and of the nasal mucosa are vertical. The author prefers a horizontal incision well toward the lower part of the lacrymal sac. (Figure 6). The horizontal incision through the nasal mu-

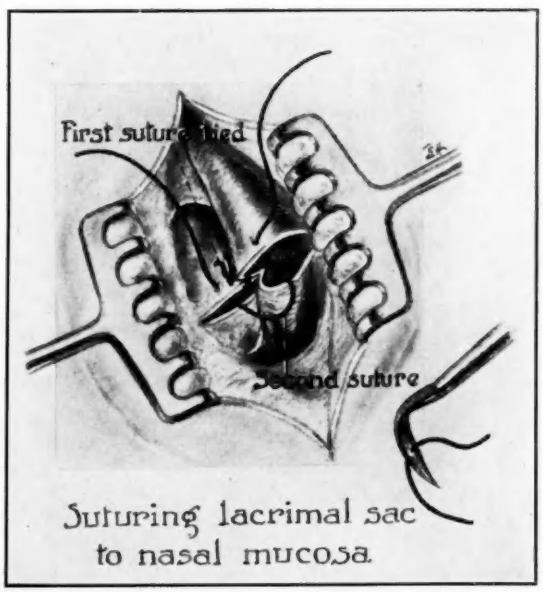


Fig. 6

cosa is more easily drawn into the bony orifice. It does not make too large an orifice from the lacrymal sac to the nasal cavity. The margins of the two layers are easily sutured by a Yankauer's needle. If the vertical incision is used, it must be well back in order that the anterior lip of the nasal mucosa is sufficient to come around the thickened bony wall. The corresponding edges of the incised mucosa of the lacrymal sac and of the nose are carefully united with sutures. One may use 000 catgut or preferably fine twisted silk which is less apt to tear the delicate membrane. Various needles have been devised to introduce these sutures. The author found the Yankauer nasal suture needles, half curved, right and left, the most satisfactory. The right needle is used for the upper sutures and the left needle for the lower sutures in the right fossa and vice versa in the left fossa. The last anterior suture is brought through the deep fascia and muscle and tied. (Figure 7). The skin is closed by interrupted sutures.

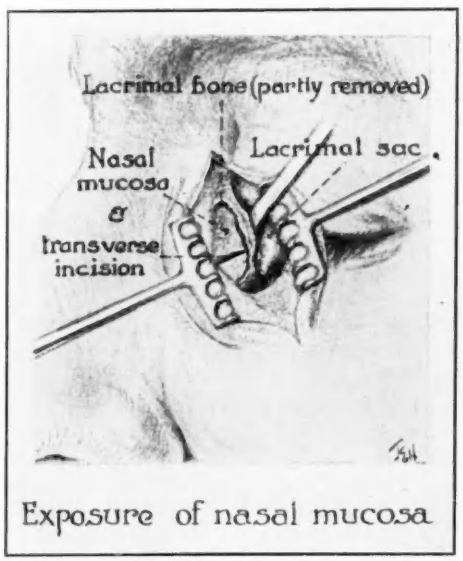


Fig. 7

White's ointment is applied freely in the cul-de-sac and over the skin incision. The eye is covered with a pad. There is some oozing from the nasal mucosa for the first twenty-four hours. The skin sutures may be removed in four or five days.

It is well to irrigate the sac the fourth day to remove blood clots. There is no other treatment necessary, unless there is considerable pus in the sac at time of operation. Under these circumstances, the sac is irrigated with normal saline for a few days. No other treatment is indicated. A complete cure will permit the passage of a solution of fluroscin from the cul-de-sac through the artificial canal to the nasal cavity.

Summary

1. Plastic dacryocystorrhinostomy does not destroy the mucous membrane of the nasal cavity nor of the lacrymal sac.

2. The artificial canal heals by first intention, without the formation of granulation tissue.

3. The physiologic function is re-established so that fluorescin passes from the conjunctival cul-de-sac to the nasal cavity.

4. Pus rapidly disappears from the sac following operation.

5. Fistulous opening from the sac closes readily after drainage is re-established.

6. No probes nor drains are necessary following operation.

7. Post operative treatment consists of removal of the skin sutures plus one or more irrigations of the sac through the canaliculus.

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DISCUSSION.

DR. E. C. ELLETT, Memphis, Tenn.: My experience with the conservative type of lacrymal sac operations aiming to secure permanent drainage into the nose by an adventitious opening, is limited to a few attempts at operations of the West type, all of which were unsuccessful. It has been my privilege to see our distinguished President perform the operation which bears his name, but for some reason I have not done it myself, possibly because it is a rather formidable procedure for one not doing intranasal surgery, and I was not sure of my ability to do it successfully.

When I was asked to open this discussion, which was to concern the Dupuy-Detemps Bourget operation especially, I wrote to Dr. Dupuy-Dutemps to find out if I could see this operation while in Paris. He kindly came to town from his holiday and arranged two operative seances for my instructions and I saw him do several cases. He has done this operation about one thousand times, and not only gets good results himself but a report by Potiquet from the Morax Clinic in the Annales d'Occulistique for June, 1929, gives the details of 234 cases, of which 92+ per cent. were successful. The operation, as I saw it, is done under local anaesthesia. There is no intranasal manipulation except the insertion of a plug wet with cocain and adrenalin, so that it can all be done by the ophthalmic surgeon who is not an intranasal operator. No special instruments are required except the needles for placing the sutures.

From seeing the operation, one can speak of the following advantages:

It is done under local anaesthesia.

It can be done by the ophthalmic surgeon, since no intranasal work is needed.

All steps of the operation are plainly visible.

It is no more formidable than excision of the sac.

No after treatment is required.

I asked Dr. Dupuy-Dutemps about failures, closure of the wound, etc. He said that when the opening into the nose tended to close, a lachrymal knife could be passed through the canaliculus straight through into the nose and the tissues between the sac and the nose cut freely up and down. This could be repeated if necessary and often would result in a cure. There are, of course, some failures.

I would like to add a little information picked up from Dr. Kronfeld at Meller's Clinic in Vienna. Meller, as we all know, is the father of the operation for the removal of the sac as it is now generally known, and of course a warm advocate of that plan of treatment. But in recent years he has had many West operations done and is extremely pleased with the result when done by a competent man; so much so that when the services of such a man are available he prefers to have this operation done to removing the sac, except in cases being prepared for intraocular operation, in which case removal of the sac gets the desired result quickly and certainly. Unless a competent man can be secured to do the West operation, the results are apparently not satisfactory.

Fig. 1. Plastic dacryorhinostomy. (After Dupuy-Dutemps.) Line of skin incision.

Fig. 2. Plastic dacryorhinostomy. (After Dupuy-Dutemps.) Lacrimal sac reflected outward. Chisel starts resection of lacrimal crest and finishes along the dotted line.

Fig. 3. Plastic dacryorhinostomy. (After Dupuy-Dutemps.) The resection of the bony window is completed. Nasal mucous membrane shown as floor in the open wound. Long incision terminating in T formation at each extremity.

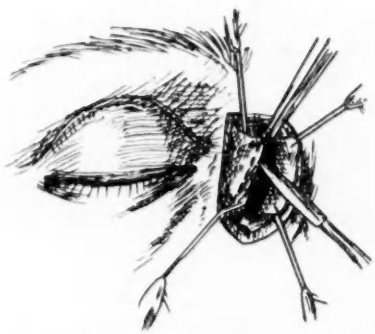

Fig. 4. Plastic dacryorhinostomy. (After Dupuy-Dutemps.) This picture shows the opening of the nasal mucous membrane made before that of the sac, according to the old method. Dupuy-Dutemps now prefers to open the sac first and clean it before opening the nasal mucous membrane.

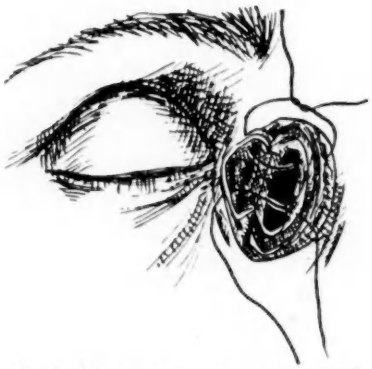

Fig. 5. Plastic dacryorhinostomy. (After Dupuy-Dutemps.) The sutures of the posterior edges are tied. Two sutures are passed into the anterior edges and through the periosteum and other subcutaneous tissues.

Dr. John A. Pratt, Minneapolis, Minn.: Dr. Knapp has given us what seems to be the last word in his method of radical operation for dacryocystitis, but we should have a conservative method to try in all cases where a probe can be passed through the lacrimal duct.

Since 1918, when Dr. W. R. Thompson presented his operative treatment of dacryocystisis, I have employed it in a modified form. I have found that the constriction is at the duct-sac end, or the duct-nasal end, and not in the center of the duct as he suggested. One should see that there are no adhesions at the duct-nasal opening, by elevating the lower turbinate. The constriction should be curetted out to at least four millimeters. Following the suggestion of Dr. Dean of Council Bluffs, in his paper of 1927, the curettment of duct and sac can be performed through an external opening into the sac, and thus avoid injury to the canaliculus. In making the external cut the sac should be filled with fluid to cut upon, the enlarging of the wound to be made with a curved Weber canaliculus knife.

After the curettment of the sac and duct constriction, as large a lead fuse wire as possible is passed through the duct into the nose, curving forward in the lower meatus. The wire is put at the top, drawn into the sac, and allowed to remain until mucous membrane has covered the curetted constricted surface. The sac can be irrigated through the external wound until it heals.

Dr. Ray K. Daily, Houston, Texas: I think Dr. Knapp's plea for the conservation of the lacrimal sac is most timely. The number of radical extirpations of the lacrimal sac still being done, make one feel that the ophthalmologists as a whole have little confidence in the procedures aiming to restore drainage into the nose; and yet a study of the literature reveals that the men who have become interested in this work and have had a sufficiently large experience to become masters of their technique have uniformly reported satisfactory results. It appears also that

the type of operation performed—an intranasal or external—is of less importance than the skill and thoroughness of the operative technique.

Our own experience has been confined entirely to the intranasal operation of Halle-West, with minor modifications; and our results have been most satisfactory; we have not encountered any of the post-operative difficulties that Dr. Knapp has mentioned; there is no need for post-operative probing or styles. Occasionally granulation tissue in the wound has to be cauterized during the process of healing, but after the patient has gotten well, so that there is no more epiphora, and secretion can not be squeezed out by pressure over the sac, we have seen no recurrence in cases that have been followed for six years. The intranasal operation has the advantage of leaving no external scar and not requiring a post-operative bandage. The results of operative procedures on the sac can be evaluated by roentgenography of the lacrimal sacs injected with lipiodol, before the operation, and again at definite intervals for several years afterwards. In cases of stricture a permanent opening above the stricture is all that is necessary. In cases of mucocele, where the sac is greatly dilated, we endeavor to destroy as much of the sac as possible, and to obliterate the mucocele; when the mucocele is not obliterated, and this can be demonstrated by lipiodol injection, the result is not so good, because secretion has a tendency to accumulate in the dilated sac. In the operation, which Dr. Knapp describes there is no attempt made to destroy a dilated sac; I should like to hear what Dr. Knapp's results are in cases of dacryocystitis with mucocele.

Dr. A. S. Green, San Francisco: I wish to put before the Section a conservative method of treating dacryocystitis, a method which we have used very successfully for over ten years. It has reduced the number of radical operations with us very markedly. If this fails, it is not too late to do something more radical.

The procedure is not entirely new, but the medicament and method of application is new. We first endeavor to wash the lachrymal passages with a solution containing cocaine, adrenalin and boric. We employ a glass syringe with a blunted Luer or a lachrymal needle, and use considerable pressure. Whether or not the solution comes into the nose, the next step consists of forcing an ointment of adrenalin, boric and lanolin into the lachrymal sac with a syringe like the one used for the solution. The patient is requested to come back three days later, and very frequently, where it was at first impossible to force through the solution it now comes through easily. The ointment is forced in again and this is done two or three times a week. We have been able to reestablish drainage by this simple means where, heretofore, we resorted to a radical operation.

Dr. F. N. Knapp, closing: Toti's original incision was approximately a half circle. It began above the internal palpebral ligament and continued below. The incision of Dupuy-Dutemps was short and included the resection of the internal palpebral ligament. I think it is better to start the incision below the ligament and continue it downward to save that structure.

The vertical incision through the lacrymal sac and nasal mucous membrane was the original procedure. In my experience unless extreme care is taken in making these incisions well back in the nasal mucous membrane, it is difficult to bring this tissue over the bony wall. For that reason, the horizontal incision is proposed in order to make this procedure easier.

I was glad to see the pictures of Dr. Daily showing the injection of the sac with lipiodol. This is a useful procedure in diagnosing any chronic condition of the lacrymal sac.

NOTE: Cuts in Dr. Ellett's discussion used by courtesy of the *American Journal of Ophthalmology*.

SOME ORIGINAL EXPERIMENTS WITH THE O'CONNOR MUSCLE SHORTENING OPERATION

JOSEPH L. MCCOOL, M. D.,

SAN FRANCISCO, CALIF.

In 1916 Dr. Roderic O'Connor of Oakland presented a paper before the Section on Ophthalmology of the American Medical Association on "A New Shortening Technique with Report of Forty-two Operations." Ten years later, in a paper presented before the American Academy of Ophthalmology and Otolaryngology, he reported the results of thirty-three shortenings of the vertical recti for the correction of hyperphoria, using a technique the same in principal but modified and improved, the outcome of ten years work with the operation.

Personal friendship, coupled with a common interest in ocular muscle problems, led me to try the operation shortly after his original paper was published and for the past twelve years I have been using it to the exclusion of all other methods. I am convinced that from the standpoint of efficiency, safety, certainty of action and the surgical principles upon which it is based, that no operation yet devised can compare with it.

Sometime ago I determined to make measurements to see if there were any more or less constant factors which could be established in securing uniform results.

Thirty-eight muscles were shortened and measurements made at the time of operating. Of this number six external recti muscles were shortened on eyes of old people who had come to operation for the purpose of having their eyes enucleated for various reasons and upon whom implantation was not considered. I might say in passing that the slight sinking in of the orbital tissue as the result of section of the externi was scarcely noticeable and certainly not objectionable to the patient.

An examination of the wet specimens should convince the most skeptical that this operation is capable of as much shortening as it is ever desirable or safe to make.

Operations which increase the influence of a muscle or muscles of the eyeball, are either tendon tucking, resection or advancement. The O'Connor operation is not an advancement—it is a shortening. The new attachment is no farther forward than the original insertion of the tendon. Its effect, therefore, is analgous to a resection or a tendino-muscular tucking. Unlike a resection, however, the tendon is never completely severed from its original insertion thereby removing two very undesirable features of resections or advancements — namely torsion from poorly placed sutures or slipping of the latter with attendant increase in the original deformity. Furthermore, the operation is capable of very definitely graduated effect to a degree possessed by no other operation with which I am familiar.

The proponents of tendon tucking claim for this operation freedom from torsion and danger of increasing the original deformity if the sutures were to slip.

However, there is one serious defect in any tucking operation which I believe is responsible for many failures, and that is the slipping of the sutures either from failure to tie them tight enough at the time of the operation, or from subsequent slipping due to sloughing.

In my opinion the greatest defect in any tucking operation is the necessity for tying the sutures in the tuck tight enough to hold the calculated amount of shortening and this very fact—the constricting effect of the sutures invites sloughing with subsequent slipping. If one side only slips you will undoubtedly have torsion with an accompanying lessening of the lateral or vertical effect desired.

Furthermore, the normal tone of the muscles at rest and the more or less involuntary contraction under innervational impulses, coupled with the pull of its opponent, all tend to exert an undesirable tension of the sutures.

So far as advancements are concerned, I have never believed that they were anything more than reenforced resections.

I am well aware of the fact that Worth, in three cases which came for reoperation, was able to pass a hook between the advanced tendon and the underlying sclera beyond the original insertion of the tendon and up to the new point of attachment. Nevertheless, I cannot convince myself that the advanced tendon and muscle do not become firmly attached to the sclera back to the original insertion of the tendon. If it were possible to operate

in such a way so as to leave undisturbed the reflection of the internal capsule surrounding the tendon after it pierces the external capsule, and keep intact the serous bursa surrounding it, it might be possible to prevent the attachment of the under surface of the tendon to the sclera. Certainly this is very difficult to do.

The technique of the O'Connor operation for muscle shortening was described fully by the author in his original paper (loc. cit.), nevertheless in order to bring out the points which I wish to emphasize in this communication I shall refer to the salient features of it. After the tendon is isolated, freed from capsular attachments and raised on a right angle hook, a small hook is inserted between the fibres of the tendon near its edge and passed back and forth from its insertion toward the muscles fibres. *(Fig. 1.)* The width of this isolated strip of tendon will vary from 2 to 3 mm. from its edge, depending upon the effect desired. This

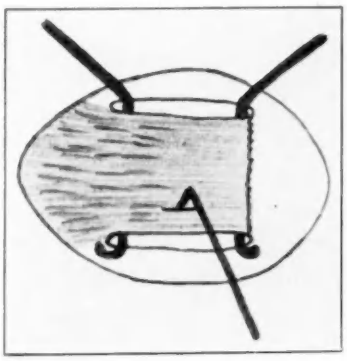

FIG. 1.—Tendon is raised on hooks and small sharp hook is inserted into the tendon preparatory to separating the lateral strips from the remainder of the tendon.

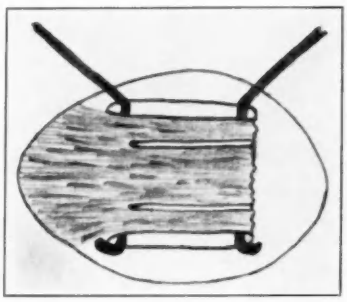

FIG. 2.—Lateral strips are separated from the remainder of the tendon.

maneuver is repeated on the other edge of the tendon. *(Fig. 2.)* What remains of the central portion of the tendon exclusive of the two lateral strips, is cut off fairly close to the insertion leaving about a 2mm. stump. This central tongue may be held out of the way temporarily by either a suture knotted around it, or it may be held by an advancement forceps. The former is preferable. *(Fig. 3.)* One lateral strip is then held lightly on a small hook and a clove hitch is thrown around it. The material used for this purpose is either fine or medium dermal (B. and B.) which has previously been threaded thru a blunt needle to facilitate the mak-

ing of the knot. By making traction on the strands of dermal sutures at right angles to the long axis of the lateral tendon strips, the clove hitch is transferred from the sutures to the tendon. In the same way another clove hitch is thrown around the other lateral strip of tendon and transferred. *(Figs. 4 and 5.)* This leaves a loop of dermal sutures between the two lateral strips. Thru this loop the central tongue is brought forward toward the corneal limbus and tied down to the original insertion of the tendon. This may be done with 000 catgut and buried beneath the

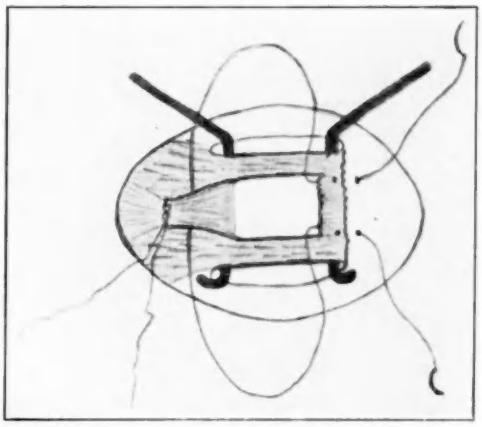

FIG. 3.—Central tongue has been cut at a point about 2 mm. from the tendon insertion and held back by suture temporarily knotted around it. At this time the fine silk or 000 catgut suture is placed. After the lateral strips are shortened with the dermal sutures the central tongue is tied down by this suture.

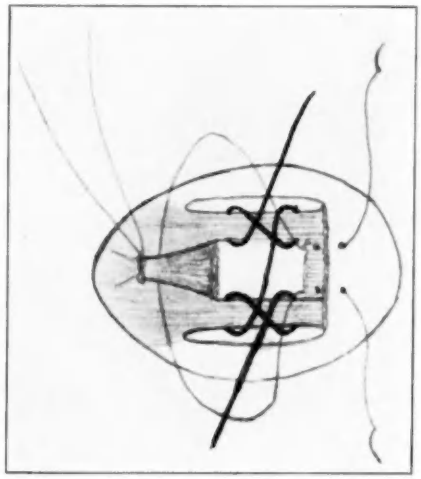

FIG. 4.—Two clove hitches of many strands of fine or medium dermal sutures are wound around the two lateral strips of tendon. They are shown here as single strands to simplify the illustration.

capsule and conjunctiva or number three twisted white silk may be used instead. In the event that silk is used (I am convinced that this is the better material for there is less reaction following its use than there is with catgut) the ends may be brought thru the capsule and conjunctiva and tied external to the latter. If the approach to the tendon is thru the meridional incision this suture will aid in coapting the wound. After the central tongue has been brought forward and tied down, traction on one end of the dermal sutures will cause the central loop to disappear and this will help to bind the central tongue down to the underlying stump. *(Fig. 6.)* One end of the dermal sutures is then cut off about 3 or 4 mm. from the knot in one lateral strip and tucked

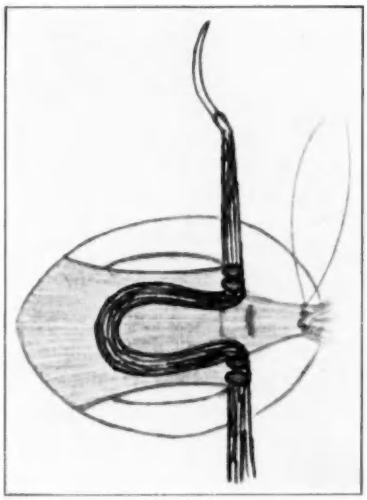

FIG. 5.—After the two clove hitches have been transferred from the dermal sutures to the lateral strips of tendon and before they have been drawn taut, there is a loop thru which the central tongue is passed and tied down lightly to the original insertion of the tendon by the suture which has already been placed.

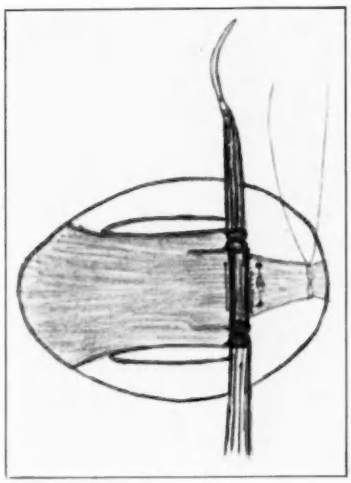

FIG. 6.—By drawing on one end of the strand of dermal sutures the loop is made to disappear and it then lies in a straight line helping to hold the central tongue in place.

beneath the capsule. The other end is brought thru a small button hole in the everlying capsule and conjunctiva. This free end should be cut off not less than two centimeters from the opening in the conjunctiva. The capsule and conjunctiva are then brought together with interrupted black silk sutures. Both eyes are bandaged for twenty-four hours. After this the bandage is removed from the unoperated eye.

In the entire operation no constricting sutures have been used nor are there any sutures under tension. The single suture used in tying down the central tongue is neither tied tight enough to cause constriction of tissue nor is it under tension, for this has all been neutralized by the shortened lateral strip. The amount of shortening is, therefore, directly dependent upon the width of the lateral strips of tendon, the number of strands of dermal suture used and their calibre. These factors were used in the measurements that I have made and tabulated and from which my conclusions are drawn. I wish to make it perfectly clear, however, that no attempt has been made to translate millimeters of shortening into arc degrees of effect.

Material used for shortening	Width of lateral strips	No. of strands of sutures	Amt. of shortening	Average shortening in mm.	Shortening per strand
Fine dermal	2 mm.	16	10 mm.		
		16	9	9.5 mm.	.59 mm.
	2.25 mm.	16	9		
		16	9	9	.56 mm.
		18	10		
		18	10		
		18	10		
		18	11	10.25	.57 mm.
	2.50 mm.	12	6	6	.5 mm
		14	8	8	.57 mm.
		16	9		
		16	9		
		16	9		
		16	9		
		16	9		
		16	9		
		16	7	8.7	.54 mm.
		18	10		
		18	11		
		18	11	10.3	.57 mm.
		20	11	11	.55 mm.
	3.00 mm.	14	11	11	.78 mm.
		16	9		
		16	11		
		16	10	10	.62
		18	10		
		18	11		
		18	11	10.6	.59 mm.
Medium dermal	2 mm.	8	8		
		8	8	8	1 mm.
	2.50 mm.	12	11	11	.91 mm.
	3.00 mm.	12	11	11	.91 mm.

PATHOLOGICAL SPECIMENS

Material used	Width of lateral strips	No. of strands	Amt. of shortening	Shortening per strand
Medium dermal	2.5 mm.	6	7 mm	1.1 mm.
		8	10 mm.	1.2 mm.
		10	10 mm.	1 mm.
		12	12 mm.	1 mm.
Fine dermal	2.5 mm.	12	7 mm.	.58 mm.
		16	10 mm.	.62 mm.

There are so many factors other than the purely mechanical ones entering into this phase of the subject that it is extremely difficult, if not impossible, to say with any degree of certainty that so many millimeters of shortening will correct so many degrees of deviation. At best it is but an approximation.

The experiments which I have made have given definite data and can be repeated by anyone. I believe that they give much more accurate information as to how much a muscle may be shortened than we could get from any resection, advancement or tucking operation.

Summary

When lateral strips of tendon measuring from 2 to 2.5 mm. in width are shortened with fine dermal, each strand will effect a shortening of .55 mm. With medium dermal the shortening per strand is 1 mm. When lateral strips of tendon measuring 3 mm. in width are shortened with fine dermal, each strand will effect a shortening of .66 mm. With medium dermal the shortening per strand is 1+ mm.

In making these experiments the original technique was used with the single exception of substituting dermal sutures for catgut.

Dr. O'Connor has made some improvements in technique which I have been using with satisfactory results, but out of deference to him I have refrained from alluding to them in this communication as I understand that he is preparing a paper on this subject for presentation at an early date.

Discussion

Dr. George F. Suker, Chicago: What one knows the least about he can talk about the most. I have never performed the operation the Doctor has described. However, it is of great interest to me and I think it is a step in the furtherance of a good cause—the muscle operation for convergent or divergent strabismus. I have some very definite views regarding the muscle operation. One is that it is largely a matter of judgment of the individual operator how much he has to do to correct the strabismus. You cannot measure your results at the time of operation in millimeters or degrees. Human flesh and blood are not wood or iron. You can in general describe a certain plan and tell why a certain operation will give a certain result, but it does not apply to the muscle operations. In the first place, I think a good many of us forget to apply the physical and mechanical laws so far as strabismic operations are concerned. You cannot transform a set of muscles which under the principle of leverage

are first class, to the second class, and have uniform action result later on. Therefore I deprecate any operation that has a tendency to so-called advancing the insertion of muscle, Dr. Worth to the contrary notwithstanding. You cannot exercise any technique so delicate without injuring the underlying sclera and the under surface of the muscle, and not have complete union exist between the advanced portion of the muscle and the sclera; therefore, you simply have, as Dr. McCool has said, a reinforcing and shortening. I have tried that time and again on dogs, and usually had complete union between sclera and muscle as a result. If it did succeed you have transposed the leverage of the second principle to leverage of the first principle, and you are bound to have more or less torsion. I believe that anyone doing a muscle operation must have regard for the insertion of the said muscles, and not alter the anatomic position. The expansion fibres you may disturb as much as you please, but the nerve impulse and muscle traction is exerted upon the entire surface of the original insertion and in cases of so-called advancement in which the muscle is still adherent to the original insertion, though its tendon has been seemingly advanced beyond, the nerve impulse and muscle traction end just the same at the original anatomic insertion.

I have no objection to any tucking operation. I think it has good value and will have an effect, but as to having 100 per cent success, or even 95 per cent success, with any tucking or resection operation, if you are expecting this, you will be sadly mistaken. After all, all your operations on muscles for the straightening of crosseyes are mainly for only cosmetic reasons. If you are true to yourself and to the patient you will know that orthoptic exercises over a period of years, after the patient has reached physiological maturity, will avail nothing in regard to the fusion of images excepting in eyes with about equal vision as in cases of alternating strabismus. Be true to yourself. The little good you can obtain is not worth the time nor the expense.

Preserve the expansion of the original insertion in a resection and accurately coaptate the muscle to the whole width of the insertion. Otherwise if you have a displacement of the muscle above or below the insertion a certain amount of torsion results; and, it is quite probable that in the operation described by the Doctor, despite the good results he has obtained, you are liable to have torsion, because the anatomic width of the muscle is not preserved. However, if you will mechanically employ the basic principle of securing actual insertion of the upper and lower portion of the muscle to the upper and lower portion of the tendinous insertion, you will avoid the ill results as nearly as is possible. No general surgeon, when he advances or shortens a muscle, ligates the same to draw it forward. Personally, I have never, either privately or in clinic, performed an advancement upon a living object, because all advancements finally are nothing but resections. Any operator who perfects his technique in any particular operation—tucking, or any resection operation, will have as good results as can possibly be obtained. It is getting the fine technique and proving your method of operation that will give you results, irrespective of the type. Furthermore, do as little trauma as you possibly can, particularly to the underlying surface of the muscle, and in addition to

the overlying surface, so as to avoid adhesions. For that reason I believe it is good policy in any resection operation not to strip the conjunctiva from the muscle, but to incorporate it with the muscle in the forceps and apply sutures through and through, and you will have no constrictions. Then, if you will pass through the sclera at the insertion of your muscle you will have no slipping of your suture. If your sutures slip it is because you have not tied them tightly or did not include some sclera at insertion, or have not drawn the muscle up sufficiently or you have been depending on one suture. You should have a supporting suture by passing it through the tendon and back of the suture in the muscle, pulling the muscle forward.

We all have our failures. I have just as many as any of you, perhaps more. But nevertheless I cannot believe that any operation that will tend to disturb and displace anatomically the insertional relationship is a justifiable operation, but any operation that employs the idea of tucking or shortening is mechanically correct.

Therefore, the operation described by Dr. McCool has my approval, as it does not disturb the original insertion.

Dr. Allen Greenwood, Boston: I did not intend to discuss this wonderful presentation until my friend, Dr. Suker, spoke and I simply want to call attention to the fact that the wonderful judgment which Dr. Suker has obtained through long practice cannot be passed on to the younger men in its entirety. It seems to me that those of us who are teaching young men, showing them how to do this operation, must give them a little of our judgment in the way of measurements. I do not think that measurement is exact in any sense of the term. This is not an exact operation. But the judgment which the older operators have got for themselves as to how much they can do is a hard thing to pass on to the younger men, unless you can give them some definite suggestions as to measurement.

Dr. Joseph L. McCool, closing: I am naturally very much pleased that Dr. Suker agrees with me regarding the value of advancement. I am inclined to disagree with him when he says that all of our good results in squint operations are cosmetic. I feel sure there are a certain number of cases which show fusion before operation. No amount of training with the amblyoscope or any other instrument will restore parallelism to the visual axes. However, after operation and training with the amblyoscope or stereoscope, not infrequently a functional as well as a cosmetic result will be obtained.

I do not know whether I quite understand what Dr. Suker means when he speaks of torsion in this operation. The very fact that the lateral strips are measured and shortened to the exact amount on both sides precludes at once the danger of creating a torsion. If I misunderstood him, I apologize.

I appreciate Dr. Greenwood's remarks, because one of the greatest difficulties that we all have is in determining how much to shorten a muscle. Every man I have asked, "How much do you cut off?" has replied, "I just use my own judgment." The amount of shortening effected by this operation, as I have specifically stated, cannot be translated into arc de-

grees of shortening, but it certainly can be duplicated by anyone else. If you have a certain width of tendon and shorten it a certain amount, you can say that you will get a certain amount of shortening, with only slight variations. Certainly such information is of value to anyone repeating the procedure.

REMOVAL OF A No. 8 LEAD SHOT FROM THE VITREOUS WITH 20/20 OR 6/6 VISION IN ONE CASE

GEORGE H. CROSS, M.D.

CHESTER, PA.

My object in presenting this paper is the hope that you will realize that eyes which have a lead shot in them *can be saved*, the shot successfully removed, and that so far, in one case of four operated upon, the patient recovered 20/20 vision following the removal, by means of the wire loop shot forceps and a double plane fluoroscope, of a No. 8 shot from the vitreous.

In the last five years I have been fortunate enough to see nine cases, within a radius of fifty miles from Chester, Pa., each one having been shot in one eye with a lead shot varying in size from a No. 8 to a B. B. shot No. 2. Dr. E. C. Ellett of Memphis, Tennessee, reported a series of thirty-seven cases from 1900-1926, which were shot in one or both eyes with various sizes of lead shot. Your imagination can conjure the number of cases that occur in the United States, many of whom should be given the opportunity to save their eye and retain its vision.

In June 1927, the first successful removal of a No. 6 lead shot from within the eyeball by this method was reported by the author in a paper read before the American Ophthalmological Society. The successful result in this case, the saving of the eyeball, was marred by the poor vision, both before and after the operation, namely, light perception.

In our endeavor to save eyes and vision, our first problem to solve when a case is presented with a small number six or eight shot in the globe, is shall we operate and attempt the removal of the shot or shall we be more conservative, take a chance and allow the foreign body to remain in the globe. The answer is—the operation *should be attempted* as quickly as possible following the injury, before the formation of exudate makes it more difficult, as we now have a method by which we can and have successfully

removed lead shot from the interior of the globe, with preservation of vision.

Before the Section on Ophthalmology of the A. M. A. in 1926, Dr. A. E. Bulson, Jr., presented a paper on "Tolerance to foreign bodies within the posterior segment of the eye." This paper involved a great deal of investigation as there are forty-one references to the reports and observation of various authorities in the text and excellent discussions of the paper. This paper deals with both magnetic and non-magnetic foreign bodies. The first conclusion is in part as follows: "It is assumed that in the majority of cases loss of vision as well as loss of the eyeball is due to inflammatory, degenerative and structural changes brought about directly or indirectly by the trauma and infection, etc.

Dr. Ancil Martin in discussion said: "A foreign body in the vitreous is a critical injury. If non-magnetic, extraction is always difficult and frequently impossible. It may result in vitreous abscess, plastic inflammation, degenerative iridocyclitis, detached retina, or phthsis bulbi, causing destruction of the eye."

Dr. E. C. Ellett presented a paper before the American Ophthalmological Society, June 1927, on "Wounds of the Eye by Projectiles of Small Caliber and Low Velocity," in which he stated in speaking of wounds of the eyeball with retention of the foreign body. "Such an eye is almost always lost as an organ of vision and even more in the case of through and through wounds is it apt to pass into a state of chronic inflammation. The situation is complicated by the fact that the small shot are not magnetic and offer only the slightest hope of removal."

The odds are against us so to speak before the case reaches our hands, nevertheless, if these cases can be treated early and promptly we can prevent many of the sequelæ and the loss of the eye, while in some instances it will be possible to save useful vision.

The most important essential point to establish before undertaking this operation is the exact location of the shot. Is it in the globe, or has it penetrated the posterior sclera and lodged in the orbital tissue, or has it entered the orbital tissue by following around the sclera.

It is quite difficult even with modern devices to localize a foreign body within two to three millimeters. This error being sufficient to place the shot either in or out of the eyeball, when in close proximity to the sclera.

In one instance, Dr. C. E. G. Shannon's case, after accurate localization of a small bird shot which was established as in the vitreous, right close to the sclera, the shot forceps were placed directly over the shot, through a scleral incision, and when directed by the fluoroscopic operator, Dr. Willis F. Manges, to move straight down I could feel the resistance of the sclera and the shot was seen in the fluoroscopic plate to be just beyond the tip of the instrument, and to move ahead of the forceps, demonstrating that the shot was not in the vitreous but had gone through the eyeball and was buried in the orbital tissues, closely against the sclera. On withdrawing the forceps and suturing the conjunctiva the shot was allowed to remain in the orbit. There were no untoward results following the operation.

In case No. 8 in which the lead shot, a B. B. was in the orbital tissue right up against the sclera we adopted a further aid in localization which was of great assistance. Holding the tip of the forceps to the side and outside the eye, it was possible on looking in the fluoroscope to line them up so that an idea of the location of the shot in the horizontal plane could be marked (by touching a spot of mercurochrome on the skin), then by holding the forceps over the eye, with the fluoroscope again lining up the tip of the forceps with the shot, its position in the vertical plane could be marked. With the lights turned on, it was possible to approximate the position of the shot. Feeling that the shot in this case was in the orbit close to the globe, I opened the eyelids and using the closed tip of the forceps as a probe was enabled to enter the wound of entrance in the conjunctival cul-de-sac and then expanding the forceps, push the tissues aside, and get a glimpse of the shot. The forceps were allowed to close and the shot was removed without any difficulty.

Case Reports.

November 12, 1924, Mr. J. W., aged 71, was shot in the left eye while rabbit hunting. He was admitted to the Chester Hospital, November 12, 1924. For this case I devised an instrument and tried to remove the shot through a scleral incision, with the help of the Roentgenologist. Using an ordinary fluoroscope, of only one position an attempt was made to get under the shot and lift it out with a single ring, bent the size and shape of the shot fashioned on the end of a needle. We were unsuccessful in our attempt and later it became necessary to remove the eyeball.

The experience in this case demonstrated the necessity of making all instruments to be used in conjunction with a fluoroscopic plate short, as the space between the eyeball and the plate, of necessity, has to be narrow to permit seeing a shadow on the plate. Long instruments run the risk of being accidently touched and damaging the internal structures of the eye, at the same time producing pain and upsetting the patient, which greatly increases our difficulties.

The next case also one in a rabbit hunter seen, November 12, 1926, was referred by Dr. C. I. Stiteler, my colleague on the Chester Hospital Staff. This was the case presented before the American Ophthalmological Society, in Quebec, June 1927. It was for this case the instrument and method, now adopted, for the removal of lead shot was devised. In this effort I was most fortunate in having the skillful assistance of Dr. Willis F. Manges who so dexterously managed the double plane fluoroscope. This was a most important factor which contributed largely to our success. Following the removal of the No. 6 shot in this case, four other cases were operated upon. In two cases the shot was found in the orbit in one the shot was removed, in the other it was allowed to remain in the orbital tissues. In the two cases in which the shot No. 8 in both instances was in the vitreous, it was successfully removed in both cases. One of which had a detachment of the retina while in the other we were able to obtain 20/20 or normal vision.

The third case in this series in which a lead shot No. 8 was definitely located in the vitreous, was Case No. 5, Miss C. G., aged 19, W. S., was struck in the face with a number of bird shot No. 8 in size, on August 1, 1928, one of which penetrated the right eyeball at 7 o'clock on the limbus cornea, just missing the crystalline lens. The localization was 10 MM. back, 8MM. below and 2 MM. to the temporal side. The patient was removed to the Jefferson Hospital the next day, when with the very able assistance of Dr. J. Farrell who operated the double plane fluoroscope we were successful in removing the imbedded shot with the wire loop shot forceps through an incision in the lower quadrant, of the sclera. This patient had a detachment of the retina which prevented a good visual result. This was a keen disappointment as the outward appearance was excellent and the eye had quieted down so promptly.

In the fourth case like the first two the patient was hunting rabbits when he was shot by another hunter, November 15, 1928, Mr. E. W. MacL., aged 24, was referred by Dr. J. Milton Griscom. Due to various complications the attempt at removal of the No. 8 shot from the vitreous of the right eye was not made until Nov. 21st, six days following the accident. Dr. Manges again took charge of the double plane fluoroscope and after the usual preliminary observations to demonstrate our ability to see the shot in the plate, an incision was made in the sclera close to the localization. After several attempts at removal and being assured by Dr. Manges in each instance that the shot was in the grasp of the forceps, on withdrawing, the shot would spring out of the forceps. We then discovered that we were grasping the shot while encased in exudate. Finally we got a firm hold of the shot and it was withdrawn. The sclera was sutured in the usual manner, atropine instilled and the eye bandaged. This case demonstrated the disadvantage of late operation, however I am pleased to report that on January 3rd, just twelve days following the operation the vision was 20/20 minus two letters and has improved since that time.

The last case with a lead shot in the vitreous makes a total of five. Mr. L. T. aged 38, came from Reading, Pa., he also was rabbit hunting when shot, November 3, 1928. It is quite a co-incidence that four out of five cases were shot while hunting rabbits. No attempt was made by his physician to remove the shot. I saw him for the first time, April 6, 1929, when his eye was a shrunken, quadrate globe, tender to touch, quite easily irritated, with the pellet of shot still inside. All that I could do in this case was to advise enucleation. He was too late.

DISCUSSION.

Dr. Allen Greenwood, Boston: This is not a very easy subject to discuss for one who has never removed a lead shot from the vitreous. Few men here perhaps will ever have the opportunity to do what Dr. Cross has done—remove a lead shot from the vitreous and have the patient retain a vision of 6/6. Most injuries by lead shot are disastrous, unless the shot enters in such a position as to make it easy of access. Many of the lead shot injuries are multiple and I would like at this time to cite very briefly an extremely interesting case—interesting from the standpoint of blindness.

Thirty years ago a young boy ten years of age came into a local hospital. He had been out hunting Thanksgiving morning with a fur cap on his head, and his companion, seeing the fur cap from the other

side of a stone wall, thought it was an animal and fired a load of No 6 shot into this boy's face. Both eyes received three or four lead shots, and, according to my judgment at the time, there was no hope of any visual results. The next summer this ten-year old was swimming in the lake near his house, and diving. In the Fall he obtained a considerable amount of money by setting traps for fur-bearing animals and selling the skins. He went through Harvard and is now a practicing lawyer in Boston.

In regard to the subject of today's paper, Dr. Cross has given us a procedure of great value for those who may have a like case. I have successfully removed a bit of copper from the vitreous by watching the blades of the forceps with the ophthalmoscope with a fully dilated pupil and was able to pass the blades around the bit of copper and remove it successfully. This vision, however, gradually left the patient's eye. Of course if the lens is cloudy this method is not available.

I doubt if many of us will ever attain the brilliant results that Dr. Cross did in his case.

Dr. E. L. Jones, Cumberland, Maryland: I think Dr. Cross is to be congratulated on opening up a new field in regard to eyes that have had penetrating foreign bodies. For many, many years I have been advocating a certain measure which I think if the Doctor would try after extraction, would give him better results as to the permanency of the cure. I think it has been the general experience that even with successful removal of foreign bodies in many cases degeneration follows. By using this method which we have followed for twenty-five years in penetrating foreign body wounds, or in cases of penetrating wounds where there is no foreign body —giving at once an injection of cyanide of mercury in sufficient quantity to cause a maximum physiologic lymphatic reaction, we can to a large degree prevent the development of sepsis which causes the loss of eyes from penetrating wounds of any nature; it also creates a tolerance for some of the non-removable foreign bodies. The tendency to slow degeneration, or to detachment of the retina, which are so apt to follow any serious trauma, can be prevented to a large extent by the administration of this injection.

Dr. George H. Cross, (closing): I wish to thank Dr. Greenwood and Dr. Jones for their interesting discussion of the paper. May I again impress upon your minds how vitally essential is the assistance of the fluoroscopic operator. Even though the operation is conducted in a dark room, Dr. Manges wears dark glasses for an hour previously to increase his dark adaption, it was his cooperation and painstaking efforts that largely contributed to our success. The forceps must be short in order that the fluoroscopic plate may be brought close to the eye so as to permit seeing the foreign body, yet not to touch the forceps. The use of the double plane fluoroscope by Dr. Chevalier Jackson and Dr. Manges in the removal of foreign bodies from the lungs first suggested its possibilities to me in the removal of these non-magnetic particles from the eye. A peculiar coincidence about these cases is that four out of the five accidents occurred in men who were hunting rabbits.

DESTRUCTIVE PURULENT OPHTHALMIA ACCOMPANYING AN ERUPTIVE FEVER WITH STOMATITIS

Report of a Case

JOHN M. WHEELER, M. D.

NEW YORK CITY

Jack Geller, age 8, a Jewish boy, was admitted to Gouverneur Hospital October 11, 1927, and the following notes were made by the house physician, Dr. C. W. Hanson:

"Patient has had the usual childhood diseases including diphtheria. He was perfectly well until two days before admission. At that time he was caught in the rain and came home feeling very badly with pains and aches all over his body. He remained sick until the day of admission when his eyes became very injected, his nose and mouth dreadfully inflamed. The mouth was causing a great deal of distress. A rash also broke out on the day of admission, two days after his first symptoms.

Physical examination—An acutely ill child, apparently suffering a great deal of pain. Head—normal shape. Face—scattered erythematous patches. Eyes—very red and much photophobia and lachrymation. Nose—slightly inflamed mucous membrane. Mouth—lips protruding and large grayish membranes over lips, tongue and pharynx. Throat—very injected and small ulcerations. Neck—negative. Chest, abdomen and legs—negative except for rash which had appearance of erythema multiforme, but did not have proper distribution. It was chiefly on chest and back, very little on extensor surface of extremities.

October 12, 1927, Skin Consultation—Found out child had been in habit of taking "Bolls Rolls" whenever sick. Made diagnosis of Erythema Multiforme, possibly caused by phenolphthalein with secondary infection of mouth and eyes (Dr. Josiah P. Thornley). Head of service at this hospital, Dr. Thos. E. Waldie of Pediatrics, believed mouth condition due to Vincent's Angina.

Several days after admission child developed typical scarletinal eruption which disappeared the next day and ulceration started on the cornea. Repeated cultures for diphtheria organisms or Vincent's Angina were negative from eyes, ears and nose. A gram positive diplococcus was always found from eye. X-ray of chest negative."

The boy was transferred to Bellevue Hospital October 23, 1927, and admitted on the eye service, where he remained until January 9, 1928, a period of seventy-nine days.

At the time of admission to the eye wards at Bellevue, the little patient had been sick two weeks. He was not prostrated and appeared in good spirits. His temperature was 99°. His appearance was pitiable because of the condition of his eyes and mouth. (See Fig. 2). The eyelids were swollen shut, with pus streaming from them. The nares were excoriated, and adherent to the lips were heavy, dark crusts and fresh blood. Scattered over the body were brownish spots unlike anything I had seen. In response to a request for a consultant from the Department of

FIG. 1.—J. G. on 17th day of illness. Brown spots on back. Scalp free from lesions.

Pediatrics, Dr. A. M. Stevens called to see the child and diagnosed the disease as that described by him and F. C. Johnson in 1922 (1) under the title, "A New Eruptive Fever Associated with Stomatitis and Ophthalmia." He said the cause was unknown and he had no definite suggestions as to treatment. The long per-

iod of observation and the importance of the disease to ophthalmologists make a report of moderate detail justifiable.

Temperature ranged from 98° to 101°. During the period of activity of the disease the temperature was near 100° most of the time. In a general way it was lower than in the previously reported cases.

Pulse ranged from 80 to 120. During the height of the disease it was usually over 100, regular and of good quality.

Respiration ranged from 18 to 26 and the patient never complained of not getting enough air in spite of the swelling of the nose and mouth.

Urine never showed albumin, casts, sugar. It never had any abnormality.

Blood examinations did not show important deviation from the normal.

Examination October 26, 1927, showed leucocytes 7200, polynuclears 78%, transitionals 6%, lymphocytes 10%, myelocytes 3%, basophiles 3%. Red blood cells showed reduction in hemoglobin. November 10, 1927, erythrocytes were 4,800,000 with 85% hemoglobin and color index of 0.9. *Wassermann* reaction

FIG. 2.—L. G. on 17th day of illness. Marked swelling of eyelids with profusion of pus. Swelling of lips, with black crusts covering part of upper lip and all of lower. Brown spots on face and chest.

was negative on October 26 and on November 7th. Chemical examination showed N P N = 35 mgm., and sugar 121.

During his stay in hospital the appetite was good, the child slept well and usually was happy. Several times he perspired profusely at night. Occasionally he said he was tired and that he had pain in the head, but apparently he was comfortable most of the time.

FIG. 3.—Case 1 of Stevens and Johnson, showing nature and extent of eruption on thirteenth day of illness.

Eyes. On admission, at the 14th day of the illness the eyelids were greatly swollen and a profusion of yellowish pus came from palpebral fissures, adhering to eyelashes, skin of lids and face. Separation of lid margins was difficult, but it was possible for the examiner to see that both corneas were almost completely ulcerated and the conjunctiva of the globe was edematous and injected. The iris could not be seen on account of corneal ulceration.

Vision O. D. Light perception.

Vision O. S. No light perception.

The secretion from the conjunctiva was persistent. It was not stringy, and conjunctival condition could not have been called membranous conjunctivitis. Bacteriological reports carried no conviction. The laboratory report of October 25, 1927, was as follows:

"Direct smear from eye: many pus cells, diploid and short-chained Gram—negative cocci, usually intracellular; no other organisms.

Culture of eye smear: blood plate shows pure culture of short chained non-hemolytic streptococci. On Loeffler's media, no

growth after 24 hours. Subculture gives mixed staphylococci, short chained streptococci and diphtheroid organisms.

Throat culture: no growth in 18 hours on Loeffler's.

October 29, 1927.

Direct Eye Smear: Gram negative biscuit-shaped diplococci, intra- and extra-cellular.

Eye smear culture: Rapid growth of small colonies of Gram negative diplococci. Gonococcus ruled out by rapidity and ease of growth, probably m. catarrhalis or pharyngis siccus. Also present, as numerous large colonies: B xerosis and "malignant" staphylococci (morphological deviations, particularly to very large organisms).

Throat culture: On blood plate—mixed culture, no organisms predominating, diptheroids occasionally present."

Ulceration of the corneas with perforation resulted in complete loss of sight in the left eye with hypotony, and reduction of the sight to light perception in the right eye. It is to be hoped that light perception will be kept in this eye, as the globe has not degenerated.

Nose. At the nares small crusts formed from excoriation, and a moderate amount of muco-purulent secretion ran from the nose. It seemed not to bother the patient much.

Mouth and lips. In the mouth, on the lining of cheeks and on the gums and tongue there were several gray patches at the time of admission and there were recurrences of patches. No bullæ were seen, but the gray spots resembled those seen in pemphigus after bullæ formation. The tongue was moderately swollen. Heavy black crusts with spots of fresh blood covered part of the upper lip and all of the lower, and there was marked swelling. The lip crusts and the mucous membrane patches persisted for weeks.

Skin. The eruption was scattered over the face, ears, chest, back, arms, forearms, hands, thighs, legs, feet. Practically the whole body was involved, but the scalp was free. There were brown patches one-half to one centimeter in diameter, very little raised, and only part of them encrusted. They did not bleed and they did not itch. There never was bleb formation. The lesions all healed without leaving scars and with almost no ultimate disturbance of pigment distribution.

When the patient left the hospital at the end of 79 days, the sight was gone in one eye and all but gone in the other. There were adherent leukomas, but nothing else to tell the tale.

Treatment. Boric acid irrigations, hot compresses, cold compresses, bichloride vaseline, argyrol, atropine, dionin were used for eye treatment. Glycerine and lemon juice were applied to lips for some time, but sterile vaseline perhaps was most helpful. If I should have to deal with another such case, I believe I should perform early canthotomy with thorough incision of the external canthal ligaments to release the eyelids, in the hope of rendering effective treatment to the conjunctiva and cornea by irrigations and antisepsis. Without relaxation of the lids proper drainage and proper treatment are impossible. *Effective eye treatment is the only urgent need.*

In a paper read before the Eye section of the American Medical Association this year, under the title of Membranous Conjunctivitis with Loss of Eyeballs, Rutherford (2) reports a case which he found difficult to classify and which may possibly be akin to those described by Stevens and Johnson and to my case.

L. H., a boy 27 months old, was admitted to the Iowa University Hospital on December 14, 1928. In October 1928, the baby had an abscess on a finger joint. He next had a coryza and a pharyngitis. About 10 days from the beginning his right eye, and 3 or 4 days later his left eye, became red and watery and the lids became swollen. The conjunctiva then became inflamed and covered by membranous exudates. He was disposed to drowsiness for days at a time.

"Examination of the right eye under general anesthesia disclosed a partial symblepharon and a corneal staphyloma; of the left, an extensive symblepharon, a healed corneal perforation and some shrinking of the organ. All four lids, none of which were now markedly swollen or tumefied, bore thick, tough grayish yellow chamois-skin-like false membranes on the tarsal surfaces. These were readily removed entirely with forceps; the underlying tissues presented a granular appearance with many bleeding points. A purulent secretion coated the nasopharynx. The sub-maxilliary and submental glands were enlarged but not hard. The temperature was elevated.

The laboratory, from numerous smears and cultures, repeatedly reported the presence of Staphylococcus aureus-hemolyticus, Streptococcus hemolyticus and Streptococcus viridans, with

one a gram-negative bacillus, but never diphtheritic or diphtheroid organisms. The false membranes consisted of the fibrinopurulent exudates which contained numerous inflammatory cells, most of which were polymorphonuclear leukocytes. The Wassermann reaction was negative.

Throughout the two months' period of observation, a seropurulent fluid was intermittently discharged from the nose; a *brownish red maculopapular eruption of varying intensity was present on the forehead, cheeks, thorax and forearms.* The department of pediatrics in several physical examinations, found nothing to account for the temperature variations, as recorded in the accompanying chart, except a persistent respiratory tract infection."

The right eye was removed, December 21, 1928, and the left, January 21, 1929. The patient was discharged, February 11, 1929; at that time both sockets were discharging a purulent material similar in appearance to that seen on admission, except that it was not so fibrinous or abundant. At the time of discharge the disease had had a duration of four months.

At a recent meeting of the Brooklyn Ophthalmological Society, Dr. John Bailey reported a case that had been recognized as belonging to the group discussed in this paper. He had called Dr. Stevens, who had agreed with Dr. Bailey that the case belonged to the class of cases described by him. I shall not give the details of Bailey's case report as he purposes publishing them soon. Dr. Bailey's paper will be a valuable one, as he had the opportunity, as an ophthalmologist, to study the patient in the early stages of the disease.

Stevens and Johnson in their paper report two cases, which they studied with Dr. L. Emmett Holt, Dr. John A. Fordyce and Dr. Warfield T. Longcope. No recognized diagnosis could be made from the symptoms and course of the eruption in these two cases, and no description of a like skin condition could be found. "Case 1 (H. S.) was a white boy, aged 8 years, admitted to Bellevue Hospital May 1, 1922, on the tenth day of his illness. There was nothing significant in the family history; he had diphtheria at 18 months, pertussis at 3 years and measles at 7 years. He had been successfully vaccinated and the tonsils had been removed at 7½ years. His general health had been good until shortly following the tonsillectomy when he was in a hospital for fourteen weeks with a septic temperature, enlarged spleen, jaundice and a blood

culture which showed staphylococcus aureus on two occasions. He recovered from this, and was apparently well for two and a half months prior to the onset of the present illness."

"Case 2 (S. M.) was a Jewish boy, aged 7 years, with a negative family history, who had been always strong and well except for measles at 4 years. He had been vaccinated successfully. Three weeks before admission, he complained of pain in the eyes and weakness. The next day the eyes were discharging, and a rash was noted on the body which the family physician called "black measles." On the third day of his illness he was taken to the Willard Parker Hospital, where he was found to have a temperature of 103 F. with signs of bronchopneumonia at the right base, a purulent conjunctivitis with great edema of the lids, and a skin eruption of which the tentative diagnosis of erythema multiforme was made. This eruption was described on the third day as being macular, hemorrhagic, with a few papules, with areas of excoriation on the face. The eyelids were edematous and pasted shut with thick pus and the mucous membrane of the mouth was practically exfoliated. The boy remained at Willard Parker Hospital for two and a half weeks, with the pneumonia resolving gradually. The temperature ranged from 101 to 104 F. for two weeks, then became irregular at a lower average with an occasional rise to 103 F. The boy was irrational during the first week, and there was great prostration, weakness and loss of flesh. In spite of instillations of boric acid solution and argyrol, corneal ulcers formed, with perforation and abscess formation in both eyes involving all the structures of the globe."

Inasmuch as Dr. Stevens was entirely responsible for the diagnosis in the case I am reporting, and inasmuch as my knowledge of the condition is entirely due to his instruction I am taking the liberty of quoting freely from his paper.

His summary is as follows:

1. Two cases have been observed of a generalized cutaneous eruption, not conforming to any recognized dermatologic condition.

2. Both cases occurred in boys, one aged 7, the other 8, coming from widely separated parts of New York City, with no possibility of contact.

3. Both cases manifested a purulent conjunctivitis, in Case 2 going on to panophthalmitis and total loss of vision, and in Case

1 responding to treatment, but leaving a severe corneal scar. The pus showed pyogenic organisms only, no gonococci.

4. A high and continuous fever was present in both cases, explainable in Case 2 by a lobar pneumonia, but in Case 1 without apparent cause other than the skin condition.

5. The eruption showed certain characteristics, identical in each case. The onset was with fever, the rash appearing on the back of the neck and chest, spreading to the face, arms and legs during a period of about eighteen days, the last lesions to appear being on the soles and palms. At this time resolution of the first lesions began.

The eruption consisted of oval, dark red to purplish macules, separated by normal areas of skin. These became in a few days raised firm papules of brownish purple, from .5 to 2 cm. in the longest diameter, without areola, and without subjective symptoms of pain or itching at any time. A few of the largest spots showed yellow, dry necrotic centers. The lesions on the forearms and shins were smaller and more thickly crowded together. No pustules or vesicles were to be seen. The scalp was at all times free of lesions; but the mouth and lips were intensely sore and inflamed. In Case 1 bullæ were noted in the mouth at the end of the first week.

After the third week resolution began in the order of appearance of the lesions. This consisted of a shrinking of the macule to a horny oval of dark brown color, with raised papery edges. From the fourth week these scales dropped off, leaving a faint pigmented area, without pitting or scarring. By the fifth week the chest, face and back were clear, except for pigmentation, while resolution and crusting were still going on on the forearms and legs. Fall of temperature coincided with the period of resolution of the skin lesions.

Diagnosis

1. A skin eruption from drug ingestion is ruled out by careful inquiry which showed that no drugs whatever had been administered in either case.

2. A skin eruption from food poisoning can hardly be considered from the entire absence of gastro-intestinal symptoms in the presence of a high and sustained temperature reaction.

2. Syphilis is to be excluded by the character of the lesions, the age of the children, the negative history, and the negative Wassermann reaction in Case 1.

4. Pemphigus was suggested, from the observation of bullæ in the mouth of Case 1. Nothing further in the course of the disease, the appearance of the eruption and its evolution gave any support to this diagnosis.

5. Hemorrhagic measles, as it happened, was the primary diagnosis of the family physician in each instance. Both children had already had measles, in Case 1 at 7 years, and in Case 2 at 4 years. While excusable at the onset, this diagnosis is quite indefensible in view of the distinctive character of the lesion as described.

6. A sepsis with generalized eruption must be considered as a possibility in spite of the negative blood culture in Case 1. This diagnosis is suggested by the history of a prior sepsis in Case 1, and might account for the temperature and course and the occurence of a pneumonia in Case 2. The superficial nature of the lesions, their character and progressive appearance over nearly three weeks and the leukopenia argue against this supposition.

7. Erythema multiforme, from some unknown toxic cause, was proposed as a diagnosis, but this is unsatisfactory from the character and distribution of the lesion, the lack of subjective symptoms, the prolonged high fever, and the terminal heavy crusting.

8. There remains the possibility that this condition represents a distinct disease which has not hitherto been recognized.

Comment—While the true diagnosis of this condition must be left an open question, the report of these two cases seems justified if only to draw attention to the serious effects to be expected unless the eyes in any similar case receive early and painstaking treatment. The boy in Case 2 is totally blind, while the other boy escapes with an impairment of vision in one eye. Furthermore, it is hoped that this report may lead to the study of other or similar cases. Quite possibly the observation of this condition in its first stages may clear up the vexed question of its etiology.

Finally we have been impressed by the striking picture presented by these cases. Here is a syndrome of dramatic onset, with fever, conjunctivitis and cutaneous eruption. The child is prostrated, the mouth and tongue are inflamed and raw, the eyelids are swollen and pus streams from the eyes. There is a course of three or more weeks of high fever, with leukopenia. The eruption, unlike any hitherto described, comes progressively, for two weeks or more, matures and resolves in horny crusts in the

order of its appearances. The temperature falls with the resolution of the skin lesions. This syndrome suggests strongly an infectious disease of unknown etiology. We believe that this condition deserves to be considered a definite clinical entity."

Here we have this pathological entity occurring in childhood with striking signs, but of great importance only because of its destruction of sight. It is a condition of interest to the pediatrician, but one of first importance to the ophthalmologist.

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DISCUSSION

Dr. C. W. Rutherford, Iowa City, Iowa: I am indebted to Dr. Wheeler, not only for his excellent presentation of an interesting case report, but also for supplying valuable diagnostic information pertaining to the case report of mine which he has mentioned in his paper. In my patient the eye and eyelid complications were the dramatic features in a disease of unrecognized etiology. My original discussion and report attempted to show that the formation of membranous exudates on the conjunctivae, with or without ophthalmitis, is not a part of any symptom complex, but only an incidental expression which might occur in any infectious process more especially where streptococci are conspicuously present. I wish to discuss my case again, and this time with more emphasis on the cutaneous eruption.

The eruption was comparable to those described in the other three cases, but it extended up into the hairy scalp. The eye conditions were evident similar, for the difference between purulent conjunctivitis and membranous conjunctivitis is one of degree and not of kind, as the history of eye complications in smallpox and diphtheria illustrates. Stomatitis was not present and no history of it was obtainable. I did not see this patient until about sixty days after the onset of the disease; it is possible that stomatitis existed at some time, for a coryza and a pharyngitis were constantly present during the two months' period of observation. However, if stomatitis is a part of the picture, it may well be a variation of the eruption on the skin and not a distinct entity.

While searching the literature for a plausible etiological classification of this patient's disease, it was found that severe eye complications had not been infrequent with diphtheria, scarlet fever, smallpox, varioloid, typhus fever, measles, impetiginous eruptions, erythema multiforme and pemphigus, as well as with the use of some drugs, such as neoarsphenamine, which often produce an exfoliative dermatitis. The temperature records support the view that the eruptions in the four cases belong in the

same category as the preceding, and that they were due to infection. One may conclude that in these cases the eye complications are only incidental; when present they merely indicate the severity of the infectious process, if needed they do not more probably represent the susceptibility of ocular and conjunctival tissues to harm from the active organisms or their toxins. The extension to the eyes is evidently metastatic in most instances.

The essential expression of the disease under discussion, if it is really a distinct disease, is the eruption on the surfaces, whether of the skin or buccal membranes. Dr. John B. Kessler, head of the Department of Dermatology, kindly examined the published reports of all four cases. He stated that he knew of nothing in the literature on dermatology with which these eruptions are comparable, and that they may constitute examples of a new disease. His diagnostic conclusions agree with those of Stevens and Johnson. He regarded the eruption as an expression of a systemic infection. Several members of the Department of Pediatrics studied my patient; they regarded the eruption as an expression of the systemic infection. While it is a dermatological problem, severe eye complications have an academic interest to ophthalmologists. Practically, there is no known treatment which will save the eyes, once they are profoundly affected; the mild cases will recover, with or without visual impairment, as the general process subsides.

The disease has given no evidence of being contagious in any stage. My case began with an abscess on a finger joint. At the same time an older brother had a similar but more extensive lesion on a hand; he attended school regularly and none of his associates became affected. After three or four days in the observation ward following admission, this patient spent about eight weeks in a ward with other children and none of them became affected, even though one of them was found blowing a mouth organ that had been used by the patient.

The ophthalmic prognosis depends mostly upon the character of the general infection, but it is modified by the relative virulence of the invading organism or the relative susceptibility of the eye tissues to involvement. The progress of these four cases was characterized by chronicity, but they were not uniform in duration. My patient became ill a year ago, and as late as last week one socket continued to discharge a little stringy pus. This demonstrates the tenacious character of the infection. The first case recorded by Stevens and Johnson went on to a mastoiditis.

The four cases under consideration were not seen by ophthalmologists until the tenth, fourteenth, twenty-first, and sixtieth days, respectively, following the onset of illness. These delays have constituted serious handicaps, for important symptoms often become masked, or are forgotten by the persons who supply the historical data. Much valuable information might be confidently expected from an ophthalmologist who has had an opportunity to observe and study a case from its beginning. I am waiting impatiently for Dr. Bailey to publish his report.

RECURRENT HEMORRHAGES INTO RETINA AND VITREOUS—CALCIUM DEFICIENCY AS POSSIBLE CAUSE

CHARLES A. YOUNG, M. D.

ROANOKE, VA.

Since the publication of Finnoff's excellent paper on this subject, I have been able to find 69 additional cases, not including the three cases here recorded.

Recurrent hemorrhages into the retina and vitreous were first described by Von Graefe in 1855. Eales in 1880 published his paper giving a very comprehensive description of this disease which often bears his name.

In recent years the following conditions have been given as the cause of recurrent hemorrhages into the retina and vitreous.

Zentmayer believes that the cause may be found in the adrenals, whether acting independently or in conjunction with tuberculosis.

Lewis is of the opinion that lysis of the intracellular cement substance and of the cell wall itself is due to a soluble protein poison usually given off from a focal infection and resulting in retinal hemorrhages. He reports two cases with abscessed teeth, both showing the presence of streptococcus hemolyticus.

Ellett, Redding, Radcliffe and Young, Finnoff and Godwin, report cases in which focal infections are given as the cause.

MacGillwray's patient had lowered coagulation time, coagulation taking place in 75 seconds.

Jeandelize's three patients all showed hyperthyroidism, a degree of renal insufficiency and arterial hypertension.

Safar, Koby, Meissner, Braun, Hanke, Finnoff and Davis report cases which they attributed to tuberculosis.

Joynt reports one case which he considered the result of syphilis.

Valude's case showed a definite reduction in renal permeability eliminating only 35 parts of phenolsulphonaphthalein instead of 63 parts in 70 minutes.

Canwright's interesting paper concerns his personal experience as a sufferer of Eales Disease and considers constipation and improper diet as the cause, although he also had several foci of infection.

Meanor's patient, a married woman 33 years of age, had intraocular hemorrhages at every menstrual period since beginning to menstruate, the hemorrhages clearing before the next menstrual period.

McCraw and Strader each report one case in which the cause was undetermined.

McCrea reports 15 cases of retinal petechiasis, not included in this paper as cases of Eales Disease, due to an exudation of blood without rupture of the vessel wall, all of these cases being the result of focal infections; ten of the cases showed rapid and marked improvement in vision following elimination of the focal infection. McCrea, furthermore, believes that the petechiasis occurring in the bladder is the result of focal infection and both are but an expression of the same disease.

Prognosis

The hemorrhages themselves clear up fairly rapidly, particularly the early hemorrhages. In Lauber's patient, a child 11 years of age, the vitreous of the left eye after an injury, was so congested with blood that perception of light was reduced to a minimum; but after a fortnight the vitreous was again so transparent that vision was normal.

Meller, speaking of the absorption of hemorrhages from the vitreous, states, "The most favorable conditions obtain in otherwise healthy eyes, in which the hemorrhages are due to a trauma which affects only a circumscribed spot, without involving the other tissue of the eye, or where a spontaneous bleeding into the vitreous occurred in circumscribed phlebitis."

After repeated hemorrhages the vision is usually lost from proliferating retinitis and detachment of the retina, and unless further hemorrhages can be prevented most of these cases will end in blindness.

Treatment

Gonzalez recommends autohemotherapy, injecting 5 c.c. of the patient's blood in the gluteral region three times a week, later, twice a week.

Grunert obtained considerable improvement in two out of four cases by paracentesis performed 8 to 16 times. After each second or third paracentesis he uses subconjunctival injection of asterol, dionin and sodium chloride. This procedure is recommended to be used in old cases only.

Lewis advises removal of foci of infection and the use of antigen-vaccine, which in his two cases presumably prevented further hemorrhages.

Joynt obtained satisfactory results with antileutic treatment in his patient who had a three plus Wassermann.

Zentmayer believes thyroid extract to be of real value in these cases.

Redding, Radcliffe and Young, Godwin and Ellett all report cases in which foci of infection were found and removed, all of the foci being in the teeth, tonsils or sinuses.

Kobey used arsenic, potassium iodide, old tuberculin, subconjunctival injections of 2 per cent to 4 per cent sodium chloride and Westerman's aminothionin pomade.

Jeandelize believes that no antihemorrhagic treatment should be neglected and that the logical treatment as suggested by his findings might consist in periodic administration of hematithyroidin and pituitary extract.

Canwright advises proper diet, relief of constipation and removal of foci of infection.

Finnoff used tuberculin in one of his cases. Davis used tuberculin in two of his cases, losing both eyes in one case and obtaining vision of 20/200 in one eye in the other case. Hanke reports good results with old tuberculin starting with 0.001 mg., and increasing 0.001 mg. every second day in conjunction with subconjunctival injections of sodium chloride solution.

Spanyol reports excellent results by puncture and aspiration of 0.5 c.c. of vitreous but does not recommend replacement of vitreous by sodium chloride as advocated by Elschnig. Aspiration is indicated only in cases where there has been no fresh hemorrhages for some time. Braun on the other hand, reports good results from aspiration followed by replacement supplemented by autohemotherapy and irradition of the spleen.

Zur Nedden believes aspiration and replacement contraindicated in Eales Disease.

Benedict advocates the use of salvarsan for its non-specific action, he also states that patients with tuberculous lesions about

the eyes often respond much better to salvarsan than to tuberculin even in the absence of syphilis.

Case Reports

Case No. 1. Mr. R. G. C., white, age 39, first seen February, 1924, complaining of a loss of vision in left eye.

Family History: No history of tuberculosis, gout, hemophilia or eye trouble.

Past History: Never suffered from constipation, syphilis or hemophilia. Had nose bleeding just before first intraocular hemorrhage and had nose bleeding as a boy, has had no nose bleeding since first intraocular hemorrhage in 1908.

Eye History: Shown graphically in table No. 1.

Eye Examination: February, 1924, V. O. D. fingers at 1 foot, O. S.=6/45. External examination, pupillary reactions and tension normal in each eye. Fundi—O. D. considerable proliferating retinitis and vitreous opacities. O. S. slight amount of proliferating retinitis and fairly dense vitreous opacities.

Patient not seen again until August 12, 1927, at which time vision in O. D. unchanged but vision in O. S. 6/12. Fundus picture much the same as in 1924, except vitreous almost clear in O. S.

[Smart read a paper before our State Society, concerning calcium deficiency in a case of scleritis, which gave me the idea of having a study made of the patient's blood calcium as all of the usual tests and examinations had proven negative.]

The blood calcium was found to be extremely low, containing but 6.5 mgm. per 100 c.c. of blood. Dr. C. D. Nofsinger of the Lewis-Gale Hospital advised the following therapy for a period of three weeks every three months: Gluco-calcium (Lilly) 5 c.c. intravenously twice a week; parathyrin (Swan-Meyers) 1 c.c. intramusculary twice a week; thyro-calx (Mulford) tablets (size No. 2) one tablet by mouth three times a day.

Dr. Chas. Graham of Wytheville, carried out the above therapy for a period of three weeks every three months.

Strange to say, the patient can tell by his symptoms of depression and malaise that his blood calcium is low. The calcium therapy almost immediately gives the patient a sense of pep and well-being, furthermore, he has had no attacks of hemorrhages since beginning the calcium therapy two years ago, as compared to eight hemorrhages in the 3½ years preceding calcium therapy.

Case No. 2. Mr. G. N., white, age 34. Patient first seen

EYE HISTORY OF CASE NO. 1

Date	Attack O. D.	Attack O. S.	Seen by	Duration	General Examination	Treatment
May, 1908	1st		Kincaid Knoxville	3 weeks	Negative	K I & dionin
June, 1908	2nd	1st	Driver Norfolk		Suspected lues	K I & Mercury
July, 1908			Savage Nashville		Negative	K I
1909			Wescott Chicago		Negative	No treatment advised
Fall 1917		2nd	Washburn Jacksonville	6 weeks	2 or 3 abscessed teeth found. Recent loss of 15 lbs. in weight.	Teeth removed K I & dionin Subconjunctival injections of Hg. cyanide.
Fall 1918		3rd	Taylor Jacksonville	6 weeks	Infected tonsils	Tonsils removed
Aug. 1919		4th	Taylor Jacksonville	6 to 8 weeks	Negative	K I & dionin
Nov. 1920		5th	Gill Roanoke	6 to 8 weeks	Ethmoids	Exenteration of ethmoids
Feb. 1924		6th	Young Roanoke	3 to 4 weeks	Negative	K I dionin and rest
Mar. 1924		7th	Graham Wytheville			
Dec. 1924		8th	Graham Wytheville	3 to 4 weeks	Negative	Arsenic, Mercury & gold
Jan. 1925		9th	Graham Wytheville	3 to 4 weeks		
Jan. 1926		10th	Shiras Cleveland	3 to 4 weeks	Positive tuberculin. 3 abscessed teeth	3 teeth extracted O. T. 10 doses. K I & dionin Hg. caynidde injections
Fall 1926		11th	Shiras Cleveland	3 to 4 weeks	Negative	Same as above except no teeth extracted.
Mar. 1927		12th	Shiras Cleveland	3 to 4 weeks	Negative	K I & dionin Old Tuberculin
Aug. 1927		13th	Young Roanoke	6 weeks	Lowered blood calcium	Thyrocalyx Parathyroid and Glucalcium

Table No. 1

June 15, 1929, complaining of a loss of vision in the left eye. Loss of vision occurred during March, 1929, this being the fourth attack. First attack, left eye, 1920; second attack, left eye, 1923; third attack in both eyes, 1928; fourth attack in left eye, 1929.

FAMILY HISTORY: No hemophilia, eye trouble, gout or tuberculosis.

PAST PERSONAL HISTORY: Acute appendix removed in 1926; no constipation, tuberculosis, lues or hemophilia. Suffered from epistaxis from 1918 to 1924, averaging about one hemorrhage every two weeks, has had no epistaxis since 1924. Sinuses, Wassermann and general examination negative, except for two abscessed teeth, which were removed June, 1929. Blood count essentially negative. Blood calcium 9.5 mgm. per 100 c.c. Coagulation time of blood from vein 6 min.

EXAMINATION: Vision without glasses: O. D. 6/4, O. S. fingers at 18 in. External examination, pupillary reactions and tension negative. Fundus O. D. shows several hemorrhages, largest of which extends along the inferior temporal vein for a distance of five disc diameters; the upper portion of the disc shows an area of proliferating tissue containing numerous small blood vessels; near the periphery below is a large atrophic area, probably the result of an old hemorrhage. O. S. very faint fundus reflex made out.

TREATMENT: Calcium therapy; gluco-calcium (Lilly) 5 c.c. intravenously twice a week; parathyrin (Swan-Meyers) 1 c.c. intramuscularly twice a week; thryo-calx (Mulford) tablets (size No. 2) one tablet by mouth three times a day; dionin 2 per cent twice a day three weeks out of every month; thyroid gr. ½ three times a day.

Case No. 3. Mr. H. W. R., white, age 26, a patient at Catawba Sanitorium for Tuberculosis, first seen February 22, 1929, for refraction; at which time he obtained vision of 6/4 in each eye with proper correction. Fundi at that time negative, except for some proliferating tissue over the right inferior temporal vein.

Next seen June 27, 1929, at which time there were about fifty hemorrhages into the retina of each eye. Some flame-shaped but most of them clump-shaped. No vitreous opacities. Vision with glasses O. D. 6/6, O. S. 6/5.

FAMILY HISTORY: No history of tuberculosis, hemophilia, gout, or eye trouble.

PAST HISTORY: No hemophilia or constipation. Is now a patient at the Sanatorium on account of pulmonary tuberculosis. No injury of either eye. Had epistaxis for four years, averaging about ten hemorrhages per month, but has had no hemorrhages for the past twelve years.

LABORATORY EXAMINATION: Sputum positive for tubercle bacilli. Wassermann, feces, urinalysis and blood examination negative. Blood calcium 11 mgm. per 100 c.c. of blood.

TREATMENT: None except the usual sanatorium treatment for tuberculosis. Potassium iodide is contraindicated on account of his pulmonary lesions and it would seem utterly useless to give O. T. to a patient who is already getting an overdose of tuberculin into his system from the active pulmonary lesions.

Of the three cases reported above I have considered deficiency of blood calcium as the cause in Case No. 1. Undetermined in case No. 2, although it might be considered the result of ab-

RESUME OF FINNOFF'S COLLECTED AND REPORTED CASES

Cause	No. of Cases	Sex			Range of Age at 1st Hemorrhage	Eye First Affected			
		M	F	Not Specified		O. D.	O. S.	O. U.	Not Specified
Undetermined	51	44	7		16 to 43	15	15	4	17
Tuberculosis	27	20	7		10 to 37	9	13	2	3
Probable T. B.	9	8	1		15 to 23	5	4		
Gastro-intestinal Constipation and Gout	8	8	0		14 to 30	0	6		2
Syphilis	5	4	0	1	18 to 33	1	2		2
Focal Infection	4	1	2	1	23 to 40	1	2		1
Disturbance of Blood and Circulation	4	1	1	2	26 to 28	1			
Menstrual Disturbance	2		2		22 to 29	1		1	3
TOTAL	110	86	20	4	10 to 43	33	42	7	28

Table No. 2

scessed teeth or again the result of lowered blood calcium the patient having only 9.5 mg. of calcium in 100 c.c. of blood. Tuberculosis would seem to be the cause in case No. 3, as all other examinations were negative and the patient has a very definite pulmonary tuberculosis.

Conclusions

1. Recurrent hemorrhages into the retina and vitreous is evidently the result of one or more of many widely different path-

RESUME OF YOUNG'S COLLECTED CASES AND THE THREE CASES HERE REPORTED

Cause	No. of Cases	Sex			Range of Age at 1st Hemorrhage	Eye First Affected			
		M	F	Not Specified		O. D.	O. S.	O. U.	Not Specified
Undetermined	17	4		13	17 to 40	1	2		14
Tuberculosis	39	5	1	33	18 to 35		3	1	35
Probable T. B.	1		1		21	1			
Gastro-Intestinal	1	1			32	1			
Syphilis	1	1			24		1		
Focal Infection	6	5	1		21 to 70	3	1		2
Disturbance of Blood and Circulation	2	2			23 to 28	1	1		
Menstrual Disturbance	1		1		33				1
Hyperthyroidism Renal Insufficiency Arterial Hypertension	3	3			20 to 25				3
Definite Reduction in Renal Permeability	1	1	0		18		1		
Total	72	22	4	46	17 to 70	7	9	1	55

Table No. 3

ological phenomena and it might well be that two or more factors are necessary, that is, a type of symbiosis.

2. Calcium deficiency should be kept in mind as a possible cause.

3. Calcium therapy seemed in one case not only to prevent further hemorrhages but very much improved the patient's general condition.

CONSOLIDATION OF FINNOFF'S & YOUNG'S COLLECTED AND REPORTED CASES

Cause	No. of Cases	Sex			Range of Age at 1st Hemorrhage	Eye First Affected			
		M	F	Not Specified		O. D.	O. S.	O. U.	Not Specified
Undetermined	68	48	7	13	16 to 43	16	17	4	31
Tuberculosis	66	25	8	33	10 to 37	9	16	3	38
Probable T. B.	10	8	2		15 to 23	6	4		
Gastro-Intestinal Constipation and Gout	9	9			14 to 32	1	6		2
Syphilis	6	5		1	18 to 33	1	3		2
Focal Infection	10	6	3	1	21 to 70	4	3		3
Disturbance of Blood and Circulation	6		1	2	22 to 29	2	1		3
Menstrual Disturbance	3	3	3		26 to 33	1		1	1
Hyperthyroidism Renal Insufficiency Arterial Hypertension	3	3			20 to 25				3
Definite Reduction in Renal Permeability	1	1			18		1		
TOTAL	182	108	24	50	10 to 70	40	51	8	83

Table No. 4

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DISCUSSION.

DR. WILLIAM ZENTMAYER, Philadelphia: It would be of interest to discuss several features of this baffling disease which manifests itself in intraocular hemorrhages, but as Dr. Young's thesis is the etiology of the condition I shall open the discussion with this thought principally in mind.

It may be recalled that Eales, in his first communication, assigned the major role to constipation, but in a subsequent paper viewed the condition as a neurosis—a vasomotor contraction of the vessels of the alimentary canal resulting in a compensatory dilatation of the systemic capillaries. The view that tuberculosis of the vessels of the retina is the underlying cause of the hemorrhages has many adherents, as has also the belief that the cause is an autotoxic one. Some writers, among whom may be mentioned Fuchs and Romer, consider the nature of the affection unknown. In a paper read before the New England Ophthalmological Society, April 20, 1920, I suggested an endocrine disturbance as the probable cause. The argument brought forward to substantiate the theory was that in view of the variability of many of the factors that have been considered as of possible etiologic importance; the characteristic picture of tuberculosis of the retinal vessels not usually found in these cases; the fairly constant age incidence, that is, adolescence, in the cases occurring in males; the physical condition of the patients as described by Eales and present in some of my cases—lassitude, low spirits, variation in local temperature, cold hands and feet—in other words, asthenia; the tendency to over size in three of my cases; and the beneficial effect of thyroid in the treatment of the disease, the thought has long been in my mind that disturbed secretion in some of the ductless glands may be an important etiologic factor. As the activities of the endocrine organs are interlocking it would be impossible probably to indicate definitely the one at fault, although the symptoms point to the adrenals as the primary one.

In connection with another hemorrhagic affection, Moret speaks of the cause as being probably an interference with the angiotonic functioning of the ductless glands, probably the adrenals. In discussing this idea with Sajous he stated that the adrenal secretion acts directly on the arterioles and sustains their tone, and further, that this function when deficient, owing to relaxation of these precapillary vessels, may be, in recurrent vitreous hemorrhages of adolescence, an indirect cause of the hemorrhages. He further pointed out that the sympathetic system is studded with adrenal medullary substance, supplied probably by the medullary portion of the adrenals, and says that "If this is true the sympathetic ocular supply must also suffer—another cause of vasodilatation probably of the choroidal vessels."

I do not wish it to be understood that I wholly reject the tuberculous cause of this disease, but I do think that some other factor must be found to explain the striking characteristics of this group of cases, and that this may be found in the endocrines, whether acting independently or in conjunction with tuberculosis. I merely put it out as a suggestion that should be borne in mind in the further study of this affection, though it is well also to remember that, to paraphrase a recent sentence of MacRobert,—the endocrine organs possess a strange proclivity to evoke suspicion towards themselves as being the source of incomprehensible ailments.

McCallum and Vogtlein attempt to explain the moderating influence in the nerve cells. The parathyroid in some way controls the calcium exchange in the body. It may be that in the absence of parathyroid secretion substances arise which can combine with calcium, abstract it from the tissues, and cause its excretion; while the presence of parathyroid secretion prevents the appearance of such bodies. The mechanism of the parathyroid action is not determined, but the result of the impoverishment of the tissues with respect to calcium and the consequent development of hyperexcitability of the nerve cells is proven. It seems that a qualitative calcium disturbance, that is, a diminution in the ionizable or electrolytic portion of the calcium in the blood, has a definite bearing on sympathetic hyperexcitability.

So that it seems probable from what has been stated that calcium deficiency brings about changes of the intercellular substance of the capillaries which favors the transudation of the blood; owing to an absence of parathyroid secretion a deficiency arises in the blood calcium and this increases the processes of transudation through alteration in the intercellular substance of the capillaries.

Friedenburg believes that in these cases we have to deal with a biologic factor of puberty. He points out that in the female at the age of puberty we have a marked thryoid action, and he believes that in the male there is exactly the same thyroid activation.

R. S. Lamb states that when the thyroid is hypersecretive there is increased loss of calcium which predisposes to hemorrhage. The views of Friedenburg and Lamb were expressed after I had read my paper but before it was published.

While the chloride is the most active of the calcium salts and is most generally used, according to Gouterman "a more efficient way of controlling the calcium balance is by using parathyroid extract, gr. 1/10, t. i. d., by mouth, and parathyroid homme (parathomone callip) subcutaneously." He states that violet ray therapy possesses the property of restoring the electrolytic portion of the calcium in the blood.

Bardenheuer, in the "Zentralblatt fur Gynokolgie," July, 1929, states that when calcium salts are given intravenously the calcium content of the blood rises abruptly at once but falls sharply again after a few moments. When given intramuscularly, however, the rise is less abrupt and remains at its maximum for several hours. Since the hemostatic action of calcium depends on its concentration in the blood, the relatively good results of intramuscular injections of calcium gluconates as compared with the intravenous injection of calcium chloride is explained.

In my own four cases the von Pirquet was positive in two, negative in one, and not recorded in one; the blood coagulation time was normal to retarded in two, slow in one, and very slow in one.

In only two of my cases was calcium used, and in both the chloride was given. In neither case was it persistently employed and the dosage was probably insufficient. It was given to influence the coagulability of the blood.

Judgment of the value of therapy in these cases is difficult because of the tendency for the vision to vary greatly. It seems too that after a time the hemorrhages spontaneously cease to recur.

Considering now Dr. Young's idea, shared by Lamb, that deficient blood calcium may explain the recurring hemorrhages, this having been present in two of the three cases recorded, and one of the patients having had no attacks of hemorrhage since the beginning of the administration of calcium two years previously. If deficient calcium is the cause of recurrent hemorrhages it becomes necessary to determine what causes calcium deficiency and how this deficiency acts.

In a paper recently read by Dr. J. I. Gouterman before the College of Physicians on "The Role of Calcium in Essential Glaucoma," he states that "Chiari and Januecks have shown that calcium salts have an inhibitory action in the processes of transudation and exudation and that they decrease the permeability of the capillaries thus preventing the oedema produced by irritating agents such as abrin."

Herbst claims that the intercellular substance in certain tissues is altered by impoverishment of blood calcium.

Dr. W. H. Crisp, Denver, Colorado: With regard to the diseases which we do not understand, it is interesting to observe the repeated swings of the pendulum from more or less prejudice in favor of one possible etiology to another. We have gone through periods in which the belief was that probably most recurrent vitreous hemorrhages were due to tuberculous disease. Now we are hearing a good deal about endocrine insufficiency.

When we speak of endocrine insufficiency we do not explain the basis of the disorder in many cases, we simply go back to a certain point. Such insufficiency is probably symptomatic of something else we have not fathomed, perhaps some chronic infection. We know that sometimes the streptococci show a hemorrhagic tendency. I suppose many of us have had the experience of having an attack of influenza and then having attacks of nosebleed as we were getting over the influenza.

Some years ago I was very much interested in a case of recurrent hemorrhages into the vitreous in a young man 27 or 28 years old, whose eyes were extremely important to him since his work was to read and relay telegraph messages. He had fairly good general health up to that time except for a vague record of an acute lung attack in his earlier days. An x-ray picture of his chest showed rather marked old lung involvement, although probably not active. After a number of hemorrhagic attacks in the fundi, with quiet intervals, we found that we had overlooked the presence of a good many enlarged glands in the lower part of his neck and in the axillary region. The case was discussed for some

time as a possible case of Hodgkin's disease, but there was not enough generalization of the glandular enlargement to make positive this diagnosis. After some time we decided to subject him to x-ray treatment for the enlargement of the lymphatic glands. The glands shrank rapidly and the patient had not, to my knowledge, although I have heard from him occasionally, had any attacks since one very shortly after starting the x-ray treatment. In this case it seems possible that the old tuberculosis was associated with a streptococcic infection which affected the lymphatic glands, and that that had something to do with the hemorrhages into the vitreous.

DR. CHARLES A. YOUNG, closing: I just wish to thank Dr. Zentmayer and Dr. Crisp for discussing my paper. I do not believe that blood calcium deficiency is the only cause of Eales' disease. Probably something else will be found to be the cause, nevertheless, the blood calcium should be studied in all cases of Eales disease with the hope of obtaining more efficient treatment.

RECIPROCAL INNERVATION OF THE EXTRAOCULAR MUSCLES

FRANCIS HEED ADLER, M.D.

PHILADELPHIA, PA.

DEPARTMENT OF PHYSIOLOGY AND OPHTHALMOLOGY
UNIVERSITY OF PENNSYLVANIA

When one voluntarily flexes the arm at the elbow two things occur—first there is sent to the biceps muscle an excitatory impulse from the motor cortex, and secondly there is sent simultaneously to the triceps muscle an inhibitory impulse. This association of an excitatory stimulus to a muscle or group of muscles with a simultaneous inhibitory stimulus to the antagonistic muscle or group was first discovered by Sherrington in 1893.[1] He found that in the case of the flexors and extensors of the limbs excitation of one group was always accompanied by relaxation of the other, and the occurrence of this phenomenon was sufficiently widespread to warrant the annunciation of a law, which he called the law of reciprocal innervation. This states that during active contraction of a muscle there occurs a simultaneous relaxation in its antagonist. "As contraction progresses in a muscle, for example an extensor, contractile activity diminishes pari passu in the antagonistic flexor." Fulton[2].

In 1893 Sherrington[3] performed an experiment which proved that this law also held good in the case of the extraocular muscles. The experiment was as follows: the third and fourth nerves were cut intracranially on one side of a monkey; for example, the right side. After several weeks had elapsed for complete degeneration of the cut nerves to take place, examination of the animal showed a complete paralysis of all of the extraocular muscles on the right side save the external rectus muscle, innervated by the sixth nerve. The right eyeball was abducted, therefore, in the position of rest due to the tone of the external rectus unopposed by any tone of the antagonists. The only voluntary motion possible in the eye was further abduction. When,

however, Sherrington placed electrodes on the cerebral cortex in the region known to elicit conjugate deviation of the two eyes to the left he found that not only did the left eye turn sharply to the left due to the pull of the external rectus muscle of this side, but the right eyeball also moved slightly from its position of abduction in towards the midline. Since all the muscles on the right side were paralyzed save the right external rectus the movement inwards of this eye could only be due to a relaxation of the tone of this muscle, and this diminution of tone in the right external rectus in connection with an increase in tonic activity of the opposite antagonistic external rectus constitutes reciprocal innervation.

The presence of reciprocal innervation in flexors and extensors of the limbs has been demonstrated many times clinically as well as in the laboratory, but there has been no satisfactory evidence presented, as far as we are aware, which demonstrates reciprocal innervation in the ocular muscles of human beings. In fact some authors deny that it exists. Wilbrand and Saenger[4] infer that it occurs in the external ocular muscles, in speaking of lateral movements of the eyes, as follows:

"In looking straight ahead on a definite distant object all the eye muscles are under slight tension (tonic innervation). When an impulse is sent to the right turners the tension of these muscles is increased while the tonus of the left turners is diminished (Sherrington)."

Further on in speaking of divergence of the eyes is the following passage:

"The impulse for relative divergence, i. e., for diminution of convergence, goes to both internal recti muscles. The relaxation of both the interni does not occur passively by a contraction of the external recti causing a stretching in the muscles but really as a positive inhibition which even is effective when the contraction of the antagonistic muscles cannot occur, for example, in peripheral paralysis, (Sherrington and Topolansky).

Tilney and Pike[5] in 1925 objected to the interpretation of Sherrington's experiments on the grounds that "by sectioning the oculo-motor nerve the paralysis of the internal rectus muscle, as well as the position of the eye in consequence of this paralysis, introduces a pathologic element into the experiment. The unopposed action of the external rectus causes a hypertonic retraction in the muscle." They point out that the only valid solution

of the problem would come as the result of investigating the tension changes in the eye muscles directly. The experiments of deKleyn[6], however, leave little room for doubt as to the existence of reciprocal innervation in the extraocular muscles of rabbits.

He studied the behavior of the ocular muscles during artificially induced nystagmus. After removing the eyeball in the decerebrate rabbit and connecting the internal and external recti muscles directly to accurate recording levers deKleyn produced nystagmus by irrigating one or other of the two labyrinths with cold water. His records show very well that when the ipso-lateral labyrinth is irrigated, a lateral nystagmus results in which the external rectus muscle contracts slowly and at the same time the internal rectus muscle relaxes correspondingly until the muscles reach the full extent of their movement, at which point the internal rectus contracts suddenly and the external rectus relaxes with corresponding rapidity.

In their paper Tilney and Pike state that an appeal to clinical experience in the field of oculo-motor paralyses should furnish abundant evidence in substantiation of Sherrington's experiment. They state, however, that one of them has never observed any inward movement in eyeballs where there is a complete lesion of the third and fourth cranial nerve nuclei. They sent out a questionnaire to a number of ophthalmologists asking for an opinion on this question. The question which was sent to twelve eminent ophthalmologists read as follows:

"In a case of complete left unilateral oculo-motor and trochlear paralysis producing divergent strabismus to the left is there any movement of the eyeball when the patient's gaze is directed toward the right?"

With one exception the replies to this questionnaire were in the negative.

The following cases are presented as evidence that reciprocal innervation does occur in the extra ocular muscles of man, and that it can be seen clinically in cases of ocular muscle paralysis if looked for. The existence of this physiological phenomenon is to be expected in the eye muscles on the grounds of efficiency of action, and were it not for the fact that its occurrence has been doubted by several authors it would appear unnecessary to lay stress on the subject.

CASE I*

On February 2, 1929, a patient, Mary N., age 47, presented herself at the Wills Hospital on the service of Dr. Baer, showing a complete bilateral internal and external third nerve paralysis on both sides and as far as could be determined, a complete fourth nerve paralysis on both sides. Her history was as follows:

Five weeks before entering the hospital while working around the kitchen she suddenly felt sharp pains in the back of her neck followed by immediate loss of vision and in a few moments by unconsciousness. She was taken to a hospital and was unconscious until that evening. After regaining consciousness there was no paralysis of other parts of the body save the ocular muscles. She spent two weeks in the hospital and on discharge her condition, as far as the paralysis were concerned, was about the same as on admission. Her distant vision, however, recovered completely as far as she could tell. Her past history is negative save for several attacks of migraine and occasional severe headaches accompanied by vomiting. Eight years ago she had a menopause and since then the headaches have been less frequent.

Physical examination showed a rather thin, slightly built female. There is a complete paralysis of the third and fourth nerves on both sides resulting in ptosis, immobility of the eyeballs save for abduction, dilated inactive pupils and paralysis of accommodation. None of the other cranial nerves were affected. Her vision was R-6/9, L-6/12.

Ophthalmoscopic examination of the right eye showed the media clear. The disc was oval, axis 15, good color, and well outlined except for a sclera crescent with an area of choroidal pigment disturbance adjacent. There was a general uncovering of the choroidal circulation throughout. The blood vessels were normal. No discreet lesions seen.

The left eye showed media clear. The disc was oval, axis 90, of good color and well outlined. No lesions were seen. The blood vessels were normal. The form fields were normal for 3/360 white and 5/360 red and blue.

Examination of the chest revealed a chronic myocarditis with auricular fibrillation and mitral stenosis. Blood pressure

*This case was presented by Dr. B. F. Baer and the author at the Section on Ophthalmology of the College of Physicians of Philadelphia, April 19, 1929.

was 116/86. Only two front teeth remained and the gums showed slight pyorrhea.

Examination of the nose and throat showed definite pus in the spheno-ethmoidal fissure on the left side. A diagnosis was made of a bilateral sphenoiditis and operation was advised. This was carried out under local anaesthesia and the mucus membrane of the sphenoid sinus on the left side was found to be acutely inflamed. The posterior ethmoidal cells on the same side were opened but no pus was seen. A few days later the right sphenoid and posterior ethmoids were opened and the same condition was found but not as severe as on the left side. X-ray of the skull and sinuses showed evidence of bilateral ethmoid and sphenoid involvement but no other changes in the skull.

Neurological examination in Dr. Weisenberg's clinic showed no neurological signs other than the complete paralysis of the third and fourth cranial nerves. They believed that the lesion was probably a hemorrhage.

Wassermann, Kolmer, and Kahn tests on the blood were negative. All these tests were carried out in view of the fact that Uhthoff (8) has insisted so strongly that bilateral oculomotor paralysis are nearly always due to syphilis. The patient later told us that a spinal fluid examination had been made for the same purpose at the hospital where she was first taken; and that this was negative. An electro-cardiogram revealed an auricular fibrillation with marked left ventricular preponderance. Examination of the urine was negative.

The patient was put on mercury rubs daily and iodides. She left the clinic approximately three weeks after her admission and showed slight beginning recovery, judged by the degree of ptosis. There seemed to be more power in both the right and the left upper lid.

During her stay in the hospital this patient showed the following phenomena:

1. Immediately on elevating the upper lids the eyes were in a divergent position, approximating a thirty degree squint. This was the position of rest probably occasioned by the unopposed tonus of each external rectus muscle.

2. If one lid only were raised that eye which was divergent could be seen occasionally making a slight inward rotation. This rotation never carried the eye quite to the midline, and no further movement of the eye inward could be elicited voluntarily.

3. With both lids elevated the patient could rotate either eye from the position of rest outward showing definite power in each external rectus. When she was asked to look to the right the right eye was further abducted and the left simultaneously moved from its abducted position in towards the midline. This was also true of the left eye when the patient was requested to look strongly to the left. At no time was the inward motion of either eye as far as the midline.

The explanation of the above phenomena we believe to be the following:

1. With both eyes closed the eyeballs seek their position of rest, which was fairly wide divergence due to the normal tonus of both external recti increased perhaps by beginning contracture of each muscle, which is known to follow the paralysis of an antagonistic muscle.

2. The movement inward of the left eye when the patient looked far to the right is due to a relaxation of the tonus in the left external rectus accompanying the contraction of the right. There is, therefore, a reciprocal innervation of the left external rectus as demonstrated experimentally by Sherrington and others.

The same explanation holds for the right external rectus when the patient looks far to the left. The only objection which might be raised to this explanation is that the internal rectus of each eye might not be completely paralyzed and that the motion of each eye inward was due to some slight action on the part of the internal recti. In Sherrington's experiments the third nerve was cut inside the skull and therefore there could be no doubt of a complete paralysis of the internal rectus. We can only judge clinically of a complete third nerve paralysis by the lack of motion in the eye and in this case we had no complete proof that the third nerve was totally paralyzed. If, however, there were action in the internal rectus, no matter how slight, it would seem obvious that the eye would be rotated beyond the midline and this, as we have stated, did not occur.

In order to rule out positively any possible action on the part of the internal recti 1.0 cc. of a 2 per cent novocain solution was injected into and along the right internal rectus muscle, and after allowing sufficient time for complete paralysis of this muscle to take place (if it were not already completely paralyzed) an examination of the ocular movements was again made. The right eye still moved in toward the midline as before when the

patient looked to the left. This proves that the movement inward could not have been due to any remaining power in the internal rectus muscle.

Although no downward movement of either eye could be elicited a positive diagnosis of a complete fourth nerve paralysis was scarcely justified since the action of the superior oblique as a downward rotator is hardly apparent save when the eyeball is in the adducted position. This being impossible in the presence of a third nerve paralysis, it was felt caution should be used in making a positive diagnosis. As concerns our present problem, however, it would make no difference whether or not the fourth nerves were paralyzed, since the superior oblique is an abductor and not an adductor, and hence could not be held responsible in any way for the inward movement of the eye.

Case II

The following patient was kindly referred for examination by Dr. Charles A. Rankin, to whom we are indebted for the following clinical notes.

A. J., male, age 23. Noticed double vision, dizziness and nausea on April 7, 1929. He first consulted Dr. Rankin on April 27, 1929, having had two weeks of osteopathic treatment. Examination at that time showed the right eye normal. There was a complete ptosis of the left upper lid and loss of all movement of the left eyeball save for abduction. The pupil was widely dilated and fixed. Accommodation was completely paralyzed. The fundi in both eyes were normal, and form fields were normal. The cornea of both eyes showed an old interstitial keratitis.

In the position of rest the right eye fixed and the left eye was divergent. If both lids were closed and the left lid elevated manually the left eyeball moved in from divergence toward the midline in an attempt to fix. With both eyes opened it could be clearly seen that the movement in of the left eye only occurred when the right eye was turned outward. No motion of the left eye inward was possible save when the right was strongly abducted but this occurred each time the patient was directed to look to the right.

He was immediately placed on K. I. and mercury and later he was placed under Dr. Klauder's care for anti-luetic treatment consisting of injections of bismuth, K. I. and mercury. His blood Wassermann was normal.

May 25, 1929, the ptosis had markedly improved but the deviation of the left eye was unchanged. At this time, however, there was beginning power in the internal rectus as evidenced by the fact that he could draw the left eye in to fix while the right eye was looking straight ahead. From this time on improvement was marked and to date the movements of the left eye in all directions are full. The pupil is still semi-dilated and shows no reaction, and accommodation is still paretic.

This patient, therefore, showed movement inward of the left eye toward the midline in spite of a complete left third nerve paralysis. No injection of novocaine was made into the left internal rectus muscle so it cannot be positively stated that this muscle was completely paralyzed as in Case I. The whole picture, however, was sufficiently convincing to regard this as highly probable and we believe that the movement inward of the left eye was due solely to a diminution in the tone of the right external rectus which comprises reciprocal innervation.

It is probable that the reason reciprocal innervation has been overlooked or even denied in these cases of ocular muscle paralysis is due to the fact that the cases have not been examined early enough. It is a well recognized fact that contracture of the unparalyzed external rectus muscle soon begins in these cases. As Fuchs[7] states, "while in a recent paralysis of the externus, the eye, when the gaze is directed straight forward, stands in the midline, it afterwards becomes drawn in more and more, and can no longer be drawn up to the median position." This is probably the reason why Case I did not show it as well as Case II which was first seen two weeks after the onset of the trouble compared with five weeks for Case I.

We wish to express our indebtedness to Dr. Grayson McCouch for his interest and for many valuable suggestions and to Mr. Samuel Fisher of Wall and Ochs for his assistance in taking the Motion pictures of these cases.

Conclusion

These cases demonstrate that reciprocal innervation of the ocular muscles does exist in man. The inward movement of one eye in the presence of a complete third nerve paralysis when the fellow eye is abducted can only be explained by an inhibition of the contractile activity of one external rectus associated with the increase in contractile activity of the opposite antagonistic external rectus.

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DISCUSSION.

Dr. Walter B. Lancaster, Boston: It seemed to me when I first read about reciprocal innervation that it was rather obvious, and I was surprised that there was any difference of opinion about it. But there are some things to be said on the other side. For instance, if you take the tendon of the flexor profundus digitorum of the cadaver and pull on it, you do not get clinching of the fist. If you want clinching of the fist, you must contract not only the flexors but the extensors at the same time. This is to fix the wrist joint and enable the flexor muscles to act to advantage. That is true not only of the fists but of all except very simple movements. Tilney and Pike tested the muscles used in taking a glass of water and raising it to the lips. They had instruments attached to the various muscles which showed that not only the protagonist muscles that were making the movement, but the antagonists were actively contracted at the same time. A football player, or any athlete, standing ready to start at a signal, stands with his muscles taut; not only the flexors that are to start him, or the extensors as the case may be, but the antagonists are taut, and when he makes a motion they all contract in properly coordinated degree. For instance, in taking a swing at a golf ball, the awkward player tightens up his muscles and tries to hit very hard; but the ball does not go very far, and the instructor says, "Do not hit so hard." That is not what he really means; what he really means is "do not keep your antagonists so taut—relax the antagonists and put all the effort on the muscle that is doing the work." The result is an easy, smooth, almost effortless, but very effective stroke. That has an application, I think, in the case of eye muscles. If I have to use my eye muscles reading on a moving vehicle, I am conscious of a good deal more effort put into fixation; I contract and tighten my ocular muscles in an effort to keep my eye fixed on the moving objects, and so I get much quicker fatigue.

It is obvious that in doing muscular work there is an advantage in relaxing the antagonists. Less energy is required to lift weight, for ex-

ample, if the muscles which do the lifting do not have to act against the resistance of the antagonists. It is only in complex motions—and practically all voluntary movements are complex—that we get well marked contraction of both protagonist and antagonist, what Tilney calls "synchronous co-contraction." The seat of control of this is in the cerebellar lateral lobes. Loss of control = asynergia = ataxia.

All methods of investigation which permit complicated movements will inevitably show co-contraction, e. g. stimulation of cortex (Tilney employed cortical stimulation); also nearly all voluntary movements involve so many muscles that synchronous co-contraction is almost always found as it was by Tilney and Pike.

It was Sherrington's great achievement that he was able to devise and carry out experiments which reduced the problem to lowest terms. He was able to isolate the simple units from which are built up the highly integrated reflexes and reactions which make up normal movement. Sherrington says "Simple reciprocal innervation must be regarded as probably a relatively artificial reaction. Experimental conditions are much less complex than are found in natural happenings. The isolated stimulus of just one whole afferent nerve while the rest of the nervous system lies plunged at the time in approximately complete quietude, is a state of things little resembling the ordinary occurrences of daily life." But the physiologist tries to reduce things to the lowest terms, and these complex movements are made up of a combination of simpler ones in which we get true reciprocal innervation.

So it seems to me the solution of the disagreement between Tilney and Pike and those who believe as they do and Sherrington and his followers is that Sherrington is not denying for a moment that most of our movements entail contraction of both protagonist and antagonist, but that reduced to the simplest terms, any muscular action is accompanied by a nervous impulse going to the antagonist and making it relax. When I have had a spasm of the flexor muscles of the toes, that is, cramp of the sole of the foot, I find it is easy to make that spasm relax, not by stretching the muscles by force applied to the toes, but by making an effort to contract the antagonist. The stimulus going through the nerves to the antagonist to contract is accompanied by a stimulus going to the other muscles to relax, and the cramping ceases. I have no doubt that is the simple form of all innervation.

Synchronous co-contraction and reciprocal innervation are both observed facts. They are not alternative explanations, only one of which can be true. Rightly understood, there is no conflict between them.

I think we should be grateful to Dr. Adler for bringing up a subject of fundamental physiological interest, because we do not often study the ocular muscles from that point of view.

Dr. Francis H. Adler, closing: I am very glad that Dr. Lancaster brought up the question of co-contraction of muscles. It is obvious that although one may have muscles contracting reciprocally it is possible to have these same muscles contracting together at the same time even though their action be antagonistic. Thus, although the biceps and triceps ordinarily contract reciprocally one can voluntarily contract both muscles

at the same time. The same thing can occur in the eye. In the course of some experimental work on the extraocular muscles of cats Dr. McCouch and I found that it was possible to change the type of action of two muscles contracting reciprocally to co-contraction. When the external and internal rectus were contracting reciprocally in a nystagmus a suitable stimulus applied to the conjunctiva producing a wink reflex caused both muscles to go into co-contraction. The fact that antagonistic muscles exhibit co-contraction at one time, therefore, does not imply that they do not contract reciprocally at another time.

ACCESSORY EFFECTS OF THE CORRECTING GLASS BY ITS POSITION BEFORE THE AMETROPIC EYE

ALFRED COWAN, M. D.,

PHILADELPHIA, PA.

The principal axis of a lens is a line, perpendicular to the plane of the lens, upon which lie the centers of the curved surfaces and all the cardinal points. This line passes through the vertices of the curved surfaces and is, therefore, the pole of the lens. The principal points are so situated on the axis that every ray which before being refracted was directed toward the first, seems, after refraction, to come from the second, and takes a final direction parallel to that which it had at first. This means (according to the theory of Gauss) that if an eye were placed at R (Fig. 1) the ray I that enters the lens in a direction toward H, the first principal point, will appear to leave the lens as if it came from H', the second principal point. This ray, in passing through the lens, cuts the principal axis in a point (C) called the optical center.

Theoretically, then, the principal planes erected at H and H' take the places of the first and second surfaces of the lens, and the actual curved surfaces are disregarded. All distances from

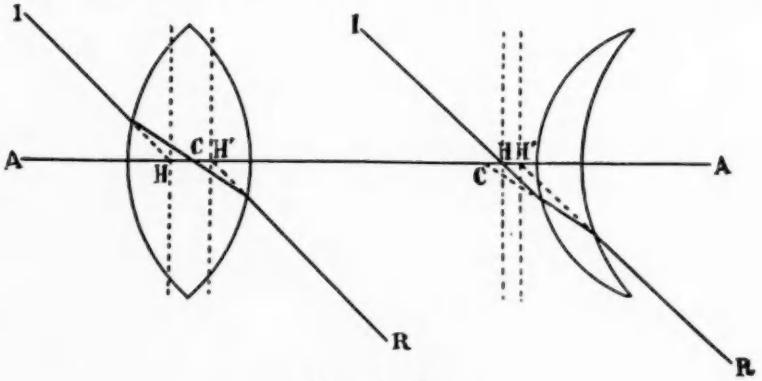

Figure I—Cowan

the lens are measured in front from H, and behind from H'. For example, the distance between the biconvex lens and meniscus (Fig 1) would be measured between the adjacent principal points, and not from the surfaces of the lenses.

It can be readily seen from the diagram that the usual assumption that a ray directed to the optical center emerges in the same direction, is not generally correct. For weak, flat lenses that may be considered infinitely thin, there will be no noticeable error, but in the modern, deeply curved lenses, especially in the higher powers, the principal points, and not the optical center, must be used to indicate the position of the lens. In any ordinary ophthalmic lens, however, we need only consider one principal point, the one that lies closest to the eye.

It should also be remembered that the positions of the optical center and the principal points vary with the shape of the lens. The practice of placing a mark on the surface of the lens corresponding to the pole, and calling this the optical center, is wrong in most cases. The optical center lies on the curved surface of a plano-convex or plano-concave lens, but in every other it lies either within or entirely outside the glass. There are two principal points and one optical center in a spherical lens, but in a sphero-cylinder there is one optical center and two principal points for each principal meridian, and the points for every other meridian of the lens lie between these two extremes, forming a line along the axis.

Therefore, in order to be correct, we shall speak of the principal point (meaning the one nearest the eye) with reference to the sagittal position of the correcting lens, and of the pole of the lens with reference to its tangential position before the eye.

The correcting lens is in the ideal position before an ametropic eye when its pole coincides with the visual line (AA, Fig. 2), its second principal point (H) coincides with the anterior principal focus (Φ) of the eye, and its posterior focus (F) coincides with the punctum remotum (P. R.) of the eye. Any variation in position introduces a more or less serious error.

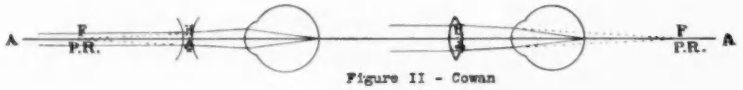

Figure II - Cowan

Prismatic Effect of a Correcting Glass

It is evidently less difficult to adjust a pair of glasses to the pupils or corneas which are easily seen than to the visual lines which are not seen. The visual line does not usually pass through the center of the pupil. This is especially true where there is a wide angle alpha. In cases of irregular or displaced pupils, from whatever cause, the visual line may not pass through the pupil at all. For this reason, generally speaking, it is wrong to adjust the lenses with the centers of the pupils. In ordinary cases, with a low refractive error, the difference between the center of the pupil and the visual line does not represent a noticeable error, but in exceptional cases proper allowance, particularly in the vertical direction, should be made. For instance, in aphakia with a strong convex lens and irregular or displaced pupils, failure to adjust the poles of the lenses to coincide with the visual lines may cause a great deal of discomfort and annoyance to the patient.

In a lens of 1 diopter the prismatic effect is 1 prism diopter for every centimeter distance from the pole. This means that in a lens of 10 diopters we have the effect of 1 prism diopter for every millimeter distance from the pole. The careful consideration of the prismatic effect of ophthalmic prescription lenses is of great practical importance.

Where the refractive error is equal in both eyes the conjugate movements of the eyes bring them into what may be called identical or corresponding points of the lenses, and the deviations are symmetrical. The conjugate upward, downward, or horizontal movements of the eyes direct them through portions of the lenses which give the effect of a pair of prisms, bases up, down, or out and in. There will be very little difficulty for the patient, but as objects will not be seen in their real position unless the visual lines pass through the poles of the lenses, it is often necessary to explain to him that objects will be apparently displaced in the direction of the apices. For example, on looking down with convex lenses the pavement will appear to be farther away, and with concave lenses it will appear to be closer than it actually is (Fig. 3). The object will also appear to move with the movement of the eyes toward the edge of the convex, and in an opposite direction in the concave lens.

Besides the apparent displacement and movement of an object, there is the magnification or elongation in the principal

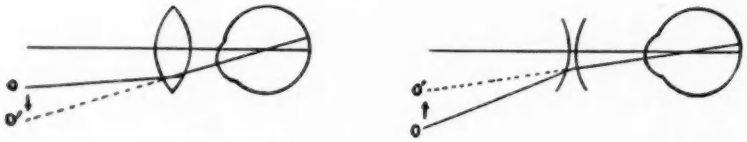

FIGURE III—Cowan

plane of the prism when the apex is turned toward the object, and a minification or shortening when the base is turned toward the object.

When the lenses are adjusted to parallelism of the visual lines they will have, during convergence, the effect of a pair of prisms bases in or bases out, depending on whether the lenses are concave or convex (Fig. 4). In the first instance—bases in—there will be less convergence needed, and in the second instance, more convergence of the visual lines. The same effect, reversed, will be achieved for distance when the lenses are adjusted for reading. It should be remembered that any alteration of the visual lines has a more or less direct influence on the judgment of distance, size and form of objects, and other phenomena associated with binocular vision.

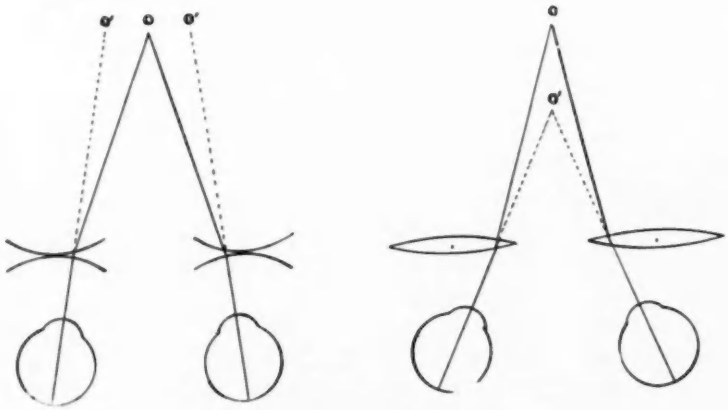

Figure IV—Cowan

In bifocals the upper and lower portions can be adjusted for their respective distances; but where the error is high and the same glasses are worn for both far and near, the problem may not be so easily solved. In some cases it will suffice to adjust the poles of the lenses to the mean distance of the visual lines be-

tween parallelism and convergence, but in exceptional cases it may be necessary to have two pairs of glasses, one for far and the other for near. In certain forms of muscle imbalance it may even be desirable to make use of this prismatic effect for one or other distance.

It need hardly be stated that prisms bases in or out can be more easily tolerated than prisms bases up and down. This effect is the result when glasses are crooked—one pole up and the other pole down—or when one eye is higher than the other. In high myopia or in aphakia, very little displacement of the correcting lenses will produce an effect equal to 2 or 3 prism diopters, a vertical displacement sufficient to cause great discomfort to the patient.

The prismatic effect of the correcting glasses becomes a serious matter sometimes in anisometropia or, in what is the same thing, astigmatism in which the principal meridians are so widely different that a relative anisometropia is produced. As already stated, where the correcting glasses are alike or nearly so, the conjugate movements of the eyes bring them into such corresponding parts of the glasses that the apparent displacements and distortions are in the same direction for both eyes; but where there is a notable difference in the strength of the lenses the prismatic effect is unequal, with consequent separation of the images and either diplopia or disturbed muscle balance. Even in these cases the deviation may not be so great that it cannot be overcome for horizontal conjugate movements, but in vertical or oblique movements the deviation cannot be disregarded. The patient should be instructed to turn the head so that he looks through the poles of the lenses. Where this cannot be done, as for instance in reading, it will be necessary to use a prismatic segment to neutralize the error.

Effect of Tilting of the Correcting Glass

When a pencil of light strikes a spherical lens in such a direction that it is not perpendicular to the refracting surface the emergent pencil will be astigmatic. The lens will be equivalent to a sphero-cylinder with the greatest power in the meridian in which it is tilted. The cylindrical effect of a tilted lens is sometimes of considerable importance in practice.

In a 4 diopter lens about .50 diopter cylinder is produced in the center by a tilting of 20°. If a sphero-cylindrical lens is

tilted in a meridian opposite to its axis the power of the cylinder is increased. On the other hand, when a sphero-cylinder is tilted in the same direction as the axis the power of the cylinder is decreased, so that in certain lenses a weak cylinder may be entirely eliminated.

Thus a pair of lenses adjusted for distance may be increased or decreased in cylindrical power when the eyes are turned down and strike the glasses obliquely in reading. The same glasses when tilted for reading introduce an astigmatic error when the patient looks straight ahead. This is the reason why it sometimes occurs that a presbyope, having been using improperly adjusted reading glasses, is uncomfortable when given the cylinder he accepted in the manifest refraction. With astigmatism against the rule, having been wearing spherical lenses, he could have found by looking obliquely through his lenses that he could make them a fairly good substitute for sphero-cylinders; or could in the same way bring a pair of too weak cylinders up to the proper strength. After having his astigmatic error properly corrected he proceeds to overcorrect his astigmatism by persisting in his old habit of tilting his glasses. Similarly, glasses placed at the proper angle for reading may introduce astigmatic errors for distance.

The cylindrical effect may be increased for one and decreased for the other, as in anisometropia where the lenses are of opposite denomination, or in astigmatism where the direction of one cylinder is widely different from that of the other. With a spherical lens for one eye and a weak cylinder for the other, it is possible that the tilting will give a cylindrical effect to the spherical lens and wipe out the cylinder in the sphero-cylinder.

Effect of the Distance Between the Eye and the Correcting Glass

It is usually assumed that an ametropic eye is corrected for distance when the posterior principal focus of the correcting glass coincides with the punctum remotum of the eye. That is to say that when this has been done the eye and the lens are the same as an emmetropic eye without a lens. We shall see that this is not exactly so.

It will be seen in Fig. 2 that since both the principal focus (F) of the lens and the punctum remotum (P. R.) of the eye lie behind the convex lens, the lens will always be weaker than

the hyperopia that it corrects, and the weaker it is the farther it must be placed from the eye. No matter how far a convex lens, whose focus coincides with the punctum remotum, is placed before a hyperopic eye an image of a distant object will be formed on the retina. A hyperope increases the corrective power of his glasses for distance by pulling them away from his eyes, and decreases their power by pushing them closer. The effect is in proportion to the strength of the lens, so that with a strong convex lens it may be considerable* If, with convex lenses, a patient continues to see clearly at a distance when the lenses are pulled away from the eyes it is an indication that he has not been overcorrected.

In myopia the punctum remotum lies in front of the eye; the posterior principal focus (F, Fig. 2) of the concave correcting lens lies in front of the lens. Here the concave lens that makes parallel rays appear to come from the punctum remotum of the eye must be stronger the farther away it is placed from the eye and, therefore, closer to the punctum remotum. A myope increases the effective power of his correcting glasses by placing them closer to his eyes, and decreases their power by pulling them away. A myope who continues to see clearly when his lenses are pulled away from his eyes is overcorrected.

So far, the assumption that the ametropic eye is corrected for distance when the posterior principal focus coincides with the punctum remotum of the eye is true; but the size of the retinal image is not the same for different positions of the correcting lens—and the size of the retinal image is always an important factor in corrected ametropia, especially in anisometropia.

Before considering the effect on the size of the retinal image by the position of the correcting glass, it might be well to briefly review the principles involved in the calculation of the size of the image in both the static and dynamic eye.

*This does not ordinarily apply to convex lenses for the correction of near vision in presbyopia. Landolt lays down the rule that in order to adapt an eye to a distance greater than double the focal distance of the convex lens, the latter will need to be weaker in proportion as it is farther removed from the eye. The reverse is true when the distance is less than double the focal distance of the lens. Thus a presbyopic emmetrope, low hyperope, or even myope who pulls his convex glasses away from his eyes in order to read, gets the effect of a weaker glass and must, therefore, be overcorrected. High hyperopes, especially aphakics, increase the effective power of their lenses by pulling them away, even for near.

The most frequently proposed method for obtaining the size of the retinal image is by the use of the proportion

$$\text{size of image} = \frac{\text{distance of nodal point from retina}}{\text{distance of object from nodal point}} \times \text{size of object}$$

But this formula is not practical for our purpose. In the static eye it cannot be applied unless we know the exact length of the eye; in the accommodating eye the changed position of the nodal points must be known.

Take Donder's eye, the simplest of all reduced eyes, in which the posterior principal focus is 15 millimeters behind the nodal point. When this eye is at rest a clear image is formed on the retina only when the object is at infinity. If the object is placed at a finite distance, such as 0, Fig. 5, the image will be formed at I. This will indicate the position of the retina in a myopic eye. The size of the image cannot be found by the above formula unless we know the length of this myopic eye. It is incorrect, in ametropia or in the dynamic eye, to use a constant distance of 15 millimeters from the retina to the nodal point (K). When the length of the eye is the same and the refractive power is increased or decreased, as in accommodation or aphakia, the distance from the retina to the nodal point is changed.

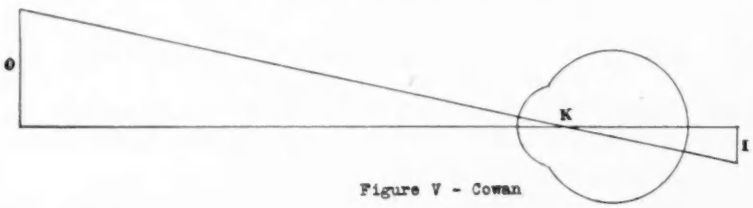

Figure V - Cowan

A better proportion for calculating the size of the retinal image is

$$\text{size of image} = \frac{\text{distance of anterior focus from eye}}{\text{distance of object from anterior focus}} \times \text{size of object}$$

The advantage of this is shown in Fig. 6, where the object is signified by O, the anterior focus of the eye by Φ and the image by I.

By this method, using a reduced eye, we can quickly find the size of the image for any distance of the object and for any length of the eye. We need only know the distance of the object and the anterior focal distance of the eye. We can also visualize the effect of the correcting glass on the size of the image.

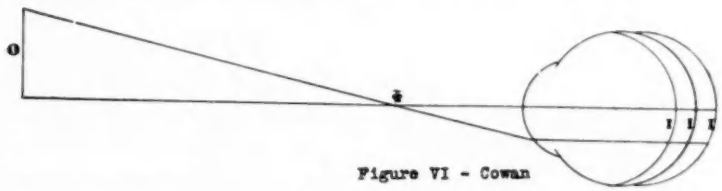

Figure VI - Cowan

If a line from one extremity of the object coincides with the principal axis of the system, and another line, from the other extremity of the object, cross in the anterior principal focus of the eye, these lines will be parallel to each other after refraction. The image, therefore, will be the same size regardless of the length of the eye. We are justified in considering all cases of ametropia as axial, excepting aphakia. The accommodating eye is considered as refractive myopia, the anterior focal distance of which we can easily find in the reduced eye.

When the ametropic correcting glass is so placed that its posterior principal point coincides with the anterior principal focus of the eye there will be no alteration in the size of the image. The reason being, as we know, that a ray directed toward the first principal point leaves the lens as though it came from the second principal point, and in a direction parallel to its first. Hence, the direction of the lines that limit the size of the image are unaffected. When the lens is placed behind or in front of the anterior focus the change in the size of the image may be considerable, depending on the distance from the focus and the amount of the refractive error.

Take, for example, three eyes: an axial myopia of 4 diopters, an emmetropic eye with a +4.28 diopter lens placed so that the principal point coincides with the anterior focus of the eye, and an accommodating emmetropic eye. We shall employ a reduced eye whose anterior focus is 16.5 millimeters in front of the cornea, and an object 25 millimeters in size, placed 250 millimeters in front of the cornea.

In the axial myopia the object is placed at the punctum remotum, a distance of 250—16.5=233.5 millimeters from the an-

terior focus. Substituting the figures in the formula, we have for the size of the image,

$$I = \frac{25 \times 16.5}{233.5} = 1.76 \text{ mm.}$$

In the emmetropic eye with the properly placed lens before it, the image would be the same size as in the axial myopia—1.76 millimeters—because the lens would render the rays parallel before entering the eye, and they would be imaged on the retina.

Our reduced eye, whose focal distance is 16.5 millimeters, has a power of 60.6 diopters. Adding the power of 4 diopters for a near point of 250 millimeters gives the accommodating eye a power of 64.6 diopters—a focal distance of 15.47 millimeters. The object is now 250—15.47 = 234.53 millimeters from the anterior focus. With these values in the formula the size of the image,

$$I = \frac{25 \times 15.47}{234.53} = 1.64 \text{ mm.}$$

It is thus seen that the image is larger for an object placed at the punctum remotum of an uncorrected myopic eye than it is when the object is placed at the same distance before an accommodating emmetropic eye. If the myopia were corrected for distance with the properly placed concave lens, so that it would be necessary to exert the same amount of accommodation as in the given emmetropic eye, the images for the two eyes would be the same.

In a young anisometrope—one eye emmetropic and the other myopic—the difference between the size of the image of an object placed at 25 centimeters from his accommodating emmetropic eye and the size of the image of an object at the punctum remotum of his myopic eye may be enough to interfere with binocular single vision. In this case a plane glass before the emmetropic eye and the proper glass before the myopic eye, will render the images the same size.

In Fig. 7 is shown the effect on the size of the retinal image when the concave lens is placed behind or in front of the principal focus of the myopic eye. In A it will be seen that when the lens is in front of the focus of the eye the ray that enters the eye as though it came from its anterior focus will have been made to diverge by the lens. This refracted ray, shown by the solid ray in the diagram, will be closer to the principal axis than the

unrefracted ray, shown by the dotted line that passes straight through the lens whose principal point coincides with the anterior focus. The image will be smaller in the myopic than in the em-

Figure VII—Cowan

metropic eye when the correcting lens is placed in front of the anterior focus. When the lens is placed behind the anterior focus, as shown in B by the solid line, the image will be larger than in emmetropia.

In myopia of high degree the correcting lens should be placed as close to the eye as possible. By doing this we not only enlarge the retinal image, but the lens will have the same effect as a stronger lens placed farther away from the eye. The largest image with the weakest lens is most desirable in high myopia. In practice the lens is usually placed at a distance of about 12.5 millimeters from the cornea, so that it is usually closer than the anterior focus of the eye, but to obtain the maximum result in extreme cases it might even be of advantage to cut the patient's lashes.

In Fig. 8 it will be seen that the effect produced by a convex lens placed behind or in front of the anterior focus of the eye is the reverse of that produced by the concave lens. Here the lens is necessarily weaker and the image larger, the farther the lens is placed in front of the anterior focus of the hyperopic eye.

Figure VIII—Cowan

One of the advantages of a meniscus, which seems to have been overlooked, is that its principal point lies in front of the anterior surface, so that it is farther away from the eye than the principal point of a flat lens of the same power and in the same position. On the other hand, the convexo-concave lens has its principal point behind the back surface, making it possible to place the principal point closer to the eye than the lens itself.

A +4 diopter lens, 3 millimeters thick, index of refraction 1.52, and a back surface of —6, has its posterior principal point about 1.66 millimeters in front of the anterior surface. When this lens is placed with its back surface 12.5 millimeters in front of the cornea its principal point is 17.16 millimeters away and the image is larger than in emmetropia. A biconvex lens of the same thickness, with its back surface in the same position, would have its principal point about 13.5 millimeters in front of the cornea—the image would be smaller than in emmetropia.

A —4 diopter lens, 1 millimeter thick, with a back surface of —6, has its principal point about .32 millimeters behind the posterior surface. The advantage of this lens over the flat form is not so great as in the meniscus, nevertheless, when placed with its back surface 12.5 millimeters in front of the cornea the principal point is only 12.18 millimeters away, and the image is larger than in emmetropia.

Where both lenses of a pair of spectacles, of the same denomination and of equal or nearly equal strength, are adjusted so that they are the same distance behind or in front of the anterior focus of the eye, there will be no discomfort to the patient; but where one of the glasses is behind and the other is in front of the anterior focus, the image in one eye will be larger than that of the other.

In astigmatism we must remember that a distortion of the image can be produced by the improper position of the lens, even though the refractive error in each meridian is corrected. For example, a convex cylinder, with its axis horizontal, and placed too far from the eye, may cause enlargement of the image mostly in the vertical meridian and make it appear elongated vertically. The reverse effect will be caused by a concave cylinder.

In mixed astigmatism it is obvious, since the curves are of opposite denomination in the principal meridians, the lens should be as carefully placed at the anterior focus as possible.

In anisometropia it is necessary to place the lenses so that the principal point of each lens either coincides with the anterior focus, or so that they are of equivalent distances from them; both in front or behind when of the same denomination or one in front and the other behind when of opposite denomination. Here, especially if one lens is convex and the other is concave, when

both lenses are too close or too far from the eyes, the image will be magnified in one eye and minified in the other.

Deeply curved lenses, when one is positive and the other is negative, must be adjusted with extreme care. In these cases will be seen one of the many advantages to be derived from the adjustment of glasses with reference to the front surface instead of the back surface, as proposed by Edward Jackson. Consider, according to Gullstrand, that the anterior principal focus of the eye is 15.7 millimeters in front of the cornea: In such an extreme case as —4 in one eye and +4 in the other, the principal point of the positive lens will be 1.4 millimeters in front of, and that of the negative lens 1.5 millimeters behind the anterior focus, if the front surfaces of both lenses are placed 15.5 millimeters in front of the corneas. This is almost perfect. But if both lenses are placed with their back surfaces 12.5 millimeters in front of the corneas the principal point of the convex lens will be 1.4 millimeters in front of, and that of the concave lens 3.5 millimeters behind the anterior focus.

There is no doubt that most of the discomfort experienced by anisometropes is caused by illy fitted glasses. There is no reason, theoretically, when the error is axial in both eyes, why these patients cannot get comfortable binocular vision with glasses. Of course, where the error is axial in one eye and refractive in the other, as in unilateral aphakia, binocular single vision is not to be considered.

DISCUSSION

Dr. W. E. Shahan, St. Louis, Mo.: This paper of Dr. Cowan's is a most important one and I hope it will receive general and thoughtful consideration. In it Dr. Cowan sets forth all the principles now causing so much confusion among ophthalmic lens designers and prescribers. He first takes up the prismatic effects of raising or lowering lenses, or moving them from side to side without tilting them, and makes a number of excellent suggestions for avoiding distressful results of these effects. He next takes up the astigmatic effects of tilting the lens in front of the eye or looking obliquely through a lens fixed in front of the eye. This is the chief defect of ophthalmic lenses and most modern improvements have been directed to reducing it to a minimum. It can be shown experimentally that a plano convex lens with the convex surface next to the eye has the greatest defect of astigmatism of oblique pencils, a biconvex lens somewhat less, a plano convex with the plane surface next to the eye still less, and that in a meniscus lens with a —6 curve next to the eye the astigmatism practically vanishes for an obliquity of 20 degrees. This is approximately the curve used in the best modern ophthalmic lenses,

such as the Toric lens on a —6 base, the Punctal, Orthogon, and Tillyer. This, however, brings us immediately to the third point discussed in Dr. Cowan's paper, that of moving the lens nearer to or farther from the eye.

In general, when a positive lens is moved away from the eye it has the effect of being stronger, and when a negative lens is moved away from the eye it has the effect of being weaker. The effective power of an ophthalmic lens is determined by the position of the principal point nearest the eye. In a biconvex lens, such as a common trial lens, this principal point is inside the lens behind the center. If measurements are made with such a lens and the physician prescribes a meniscus or other modern lens so as to avoid the evil effects of artificial astigmatism, he virtually moves the lens away from the eye and makes it stronger, so that his patient will be over-corrected. This is due to the fact that in the meniscus lens the principal point has moved forward until it is outside the lens and in front of its front surface. Some text-books meet this problem by simply stating that strong Toric lenses cannot be used. Such a statement of course is based on lack of knowledge of optical principles. Some manufacturers meet it by reducing the effective powers of their Toric lenses to that of common trial case lenses, in which case the neutralization test fails and confusion results. The best solution seems to me to be the use of trial cases in which the lenses are of average size and as nearly as possible of the same design as the patient is actually going to wear.

Dr. Cowan also shows that the optimum position of the lens in front of the eye is that in which the second principal point coincides with the anterior principal focus of the eye. When in this position the correcting lens simply moves the image forward or backwards along the visual axis without changing its size. The weakness of this lies in the fact that there is no practical way of finding the anterior principal focus in any given eye. It is probably not the same in any two eyes and is constantly fluctuating in an accommodating eye, and may be 24 mm. or more in front of the cornea in an aphakial eye. Dr. Cowan meets this weakness by placing the second principal points at equivalent distances from a theoretical anterior principal focus.

Dr. Edward Jackson, Denver, Colorado: Many of our difficulties in the prescription of lenses and getting the effect we want to the patient, arise from our uncertain or defective knowledge of these optical effects to which Dr. Cowan has called our attention. At times we have all fallen back on the resource that a human organism can adapt itself largely to its environment. We should try to see what has been natural to the patient and what is the best glass to be provided. In all cases, when we materially change the refractive conditions of the visual organ, there is necessary a certain amount of adaptation. If we will think of these problems with an understanding of the general principles which Dr. Cowan has so admirably placed before us, we will make it easier for the patient as to his problem of getting used to his glasses. Perhaps the most obvious and important lesson from this paper is that these points in

physical optics can be simply presented. It has brought diagnostic help for a class of difficulties that all have encountered who prescribe glasses, or who have to wear glasses of decided strength. It shows that these difficulties, which may still exist to some extent, can be reduced to the minimum by selecting the optimum glass and the optimum position for the glass. Also, by a little preliminary explanation based on an understanding of the mathematics of the problem, it is possible to make it very much easier for the patient, by gaining his confidence at the outset, which will result in strengthening his efforts to rapidly accustom himself to the new conditions of vision.

Dr. Sidney L. Olsho, Philadelphia, Pa.: I feel that to get the full effect of Dr. Cowan's paper one should have an opportunity to stay with him a number of hours instead of the few minutes he has been able to give us on this important subject. It is one that interests me very greatly and has for a number of years. Some of the problems which are brought out by Dr. Cowan I have, during that time, endeavored to reduce to a practical standpoint in harmony with scientific teaching. If we are informed in these principles there will be no outraging of them in the conduct of our refractive methods.

The problems discussed have been before the designers of ophthalmic test lenses for many years and the difficulties have been appreciated. There are many which are perhaps impossible of complete solution. One of the earliest changes made in test lenses was to make the convex test lenses of a slightly different focus from that which they are marked. The error because of thickness was compensated for, simply by making the convex test lenses of such a focus that they would be neutralized by the negligibly thin concave master lenses. The concave lenses were marked true to focus. The convex spherical test lenses were true as regards back focus. If we prescribed a +4. lens from the test case and received from an informed optician a lens which had the same back focus, we received almost identically what we prescribed were it a Biconvex, Plano convex or Meniscus lens, providing that in each of these instances the power were measured from its back surface and distances to cornea corresponded.

Something has been said about tilting the lenses and the effect thereof. In my refractive work I endeavor to position my test lenses on a line parallel to the upper and lower thin bony margins of the orbit. I do not admit that the position of my lenses is a tilted one although it is necessary to tilt the trial frame temples sufficiently to bring the planes of the test lenses parallel to the bony orbital aperture, and although subsequently it also is necessary to tilt the spectacle temples sufficiently to bring the planes of the ophthamic lenses parallel to the same upper and lower thin bony margins of the orbit. This position as nearly as I have ever been able to judge is the only one which places the lens planes at right angles to the visual lines when the eyes and head are in the primary position neither elevated nor depressed.

If the trial frame lenses are not so positioned they are really out at the bottom and are practically tilted upward. A refractive test of a hyperope with the lenses so placed, out below, might result in a prescription to include a cylinder at 180 degrees, in a hypothetical case for which my

prescription would call for a simple sphere. An important thing in my opinion is to insure agreement in position as between the test lens and the ophthalmic lens.

DR. E. E. BLAAUW, Buffalo, New York: I only want to add just one thing regarding the last sentence: "Of course where the error is axial in one eye and refractive in the other, as in unilateral aphakia, binocular single vision is not to be considered.

That is not exactly right. I remember as a young student that Donders had operated a patient and when he got normal vision in the operated eye as in the other eye, that was not operated, that man was so distressed that he wanted Donders to remove the good eye!

About fifteen years ago the Zeiss people undertook to help out this condition with special spectacles, for often if there is a condition of aphakia there is some binocular single vision. There is a certain amount that can be measured with Brooksbank James' instrument showing the distance of the white thread.[1] Before we refract we should examine the condition of the binocular single vision with every case of pronounced anisometropia, because if it is not present you may get a correction that gives the patient much discomfort and you cannot overcome this incompatibility in later life.

1. "Binocular Single Vision." Trans. Sec. Oph. A.M.A., 1917.

DR. ALFRED COWAN, closing: I would like to say this, that while it is a pretty good thing to know the optical theories and principles involved in all cases, in many instances it suffices merely to know them. I do not believe in carrying exactitude to such extremes that it seems ridiculous, but I think we should know these principles, first of all so we can explain to our patients, even though we cannot help them, some of their problems and certain optical defects in an ophthalmic glass that we cannot overcome. On the other hand, it is the extreme case in which we need to take advantage of every principle involved, and it is the extreme case that becomes the important one.

Concerning the focal power of the finished lens and the trial case lens: some time ago I collected a few lenses, measuring the focal power from the front surface through the lens to its focal point, and I found that the difference in focal power for the biconvex lens, the plano-convex lens, and the deep meniscus lens was so slight that they are interchangeable. That may seem radical, but it is true. If you take the distance from the front surface of the trial case lens to the patient's face and insist on that distance of the front surface in your finished glass, I do not think you need consider the shape of the glass; the difference is so slight that it is negligible.

So far as the best position of the glass is concerned, I think it is a good general rule to place the glass as far away from the patient's face as possible in a hyperope, except an aphakia, and as close to the patient's face as possible in a myope. In some cases, as I have already said, it may be necessary to clip the patient's lashes in order to place the lens close enough.

Just a word about Dr. Olsho's principle of adjusting the glasses. It is very well to adjust the glasses at an angle for central vision, that is, to have your finished lens agree in obliquity with the obliquity of the trial case lens in the center. Of course, you have a tilting with any glass when you look up, down, out or in, even though the planes are perpendicular to the visual line, but when they are adjusted perpendicular to the visual line you have the same tilting below as above. But by Dr. Olsho's method you have an outward tilting above and an inward tilting below, and I think that might be confusing to the patient.

What Dr. Blaauw has said I suppose is true. I have great respect for his knowledge. What he said about binocular single vision is right. You should find out if the patient has good binocular single vision before attempting to correct anisometropia, and where the patient has no binocular single vision you are better off not to correct the most defective eye.

POST TRAUMATIC OCULAR TUBERCULOSIS

NELSON MILES BLACK, M. D.
F. HERBERT HAESSLER, M. D.

MILWAUKEE, WIS.

Posttraumatic ocular tuberculosis is an infrequently observed occurrence. Our own experience is limited to two cases, one of which is not unequivocal and a search through the literature resulted in a rather meagre yield.

CASE 1

Mrs. D. H., age 70 years, was first seen in October, 1926. Aside from incipient cataract with well marked radial opacities in the lenticular cortex, both eyes were free from abnormality. She was given Dionin and Potassium Iodide solution and by November, 1927, the lenticular opacities had advanced to a degree to make operation seem advisable. A combined iridectomy and extraction was done under local anesthesia on November 15, 1927. The eye healed satisfactorily. On discharge from the hospital November 28th it was grossly normal with only slight hyperemia and a white mass presumably of lens cortex in the pupillary area. A slitlamp examination on this day revealed a shallow anterior chamber, with a large white mass, possibly exudate, interposed between the center of the cornea and the vitreous which presented in the pupillary area. The vitreous bulged so that it was nearly in contact with the cornea and was heavily sprinkled with large and small pigmented deposits. The iris was adherent to the vitreous and had several widely dilated bloodvessels visible in its tissue. With correcting lens the eye had a visual acuity of 6/10.

In January, 1928, the eye was still hyperemic, the media somewhat hazy and the visual acuity 6/12. In February the eye was more red and a rather thick plastic exudate filled the entire coloboma of the iris. Deep corneal vascularization was observed and the dilatation of the iris vessels was striking. She was seen occasionally during the year (she came from another city). In October, 1928, we noted that the iris presented a picture typical of tuberculosis. The cornea was opaque and vascularized in the

upper quadrant and there were very numerous unpigmented post corneal deposits varying from the finest granules to large lardlike masses. The anterior chamber was shallow. The iris was firmly bound to a pseudomembrane of plastic exudate which filled the pupillary area and by its contracting displaces the iris upward. Only two small openings into the posterior chamber could be found. Distributed through the iris stroma were nine pearly grey translucent, protruding masses, chiefly peripherally placed though one or two were in the pupillary zone of the iris. Many of them were surrounded by dilated bloodvessels. Krueckmann's plate in the Graefe-Saemisch Handbuch illustrates this clinical picture precisely. An Internist who examined her generally at this time made a diagnosis of active pulmonary tuberculosis, based chiefly on the presence of fine rales over the upper right lobe and a loss of one fifth of her bodily weight. There was little rise in temperature. The Wassermann reaction was negative.

In August, 1929, the eye was free from hyperemia, the posterior surface of the cornea had become practically free from deposits, and the lesions interpreted as tubercles had begun to undergo retrogression. Most of these were replaced by atrophic spots in the iris stroma.

To be sure, the iris picture here is not pathognomonic of tuberculosis. There is probably no form of tissue reaction in the iris which can definitely be traced to only one particular etiological agent. However, in the presence of an active tuberculous process in the lung, a negative Wassermann reaction, and an iris presenting a clinical appearance which has until recently been generally considered as pathognomonic of tuberculosis, it seems probable that the iritis in this case was tuberculous.

CASE 2

A child seen at the Milwaukee Children's Hospital is one of bilateral iritis following trauma to one eye. The child ten years old was struck in the eye with a piece of ice, without rupture or penetration of the bulbus. Two months later his vision was greatly decreased and he entered the hospital with a bilateral uveitis and partial cataract. The iris in each eye was swollen, there were many synechia and a thick membrane of plastic exudate in the pupillary area, as well as numerous thick unpigmented post corneal deposits. Tuberculosis was suspected but the internist could find no evidence of a tuberculous focus elsewhere in the body. There was no rise of temperature, no reaction to an injection

of one milligram of old tuberculin. A satisfactory X-Ray plate showed no evidence of intrathoracic tuberculosis. Liver and spleen were not palpable and the Wasserman was negative. Despite the vigorous local treatment with nonspecific protein therapy, administered during a long stay in the hospital, the eyes grew steadily worse. One year after the injury, translucent, sharply defined greyish swellings were seen in the iris. The appearance of the iris was in every way as characteristic of tuberculosis as in the first case described. Of course the negative Wasserman reaction does not entirely rule out syphilis but it surely is more significant than the negative findings in a search for tuberculosis. It is also impossible to say whether the iritis is secondary to a form of infection neither tuberculous nor syphilitic. The possibility of sympathetic ophthalmia must be considered but in the absence of evidence of perforation of either eye ball this thought has scant support.

In his collective review in the Graefe-Saemisch Handbuch Wagenmann mentions four cases similar to our second one, but none like the first. Dr. Jonas Friedenwald was kind enough to send us the following note on a case observed by him:

"The patient was a young man who had recurrent hemorrhages in the vitreous over a period of a year or more. Cataract eventually developed and was extracted (not by me). Following the extraction a severe uveitis developed and the eye ball had to be enucleated. The histological examination showed a conglomerate tubercle in the flat part of the ciliary body which had discharged into the vitreous. There were miliary tubercles on the surface of the iris, on the under surface of the cornea, in the iris stroma, about the canal of Schlemm, in the sclera at the limbus and in the episcleral tissue. There were also miliary tubercles in the choroid far anteriorly and about the retinal veins. The retina was completely detached; lens absent; beginning phthisis bulbi. The case presents a beautiful confirmation of Dr. Verhoef's theory that tuberculosis kerato-iritis is the result of a discharge of tubercle bacilli into the vitreous from a localized caseous lesion in the ciliary body or farther back in the eyeball."

F. W. Block reports his observations on a patient whose eye had been injured by a red hot splinter of iron. One month later he entered the clinic with a severe iritis with hypopyon and numerous synechia. The Cornea was hazy and had a linear scar. The eye healed under local treatment but a linear iris defect was

discovered under the corneal scar. No evidence of intraocular foreign body nor syphilis was found. The iritis recurred and a diagnosis of tuberculosis was made on the basis of the reaction to tuberculin. Block found no similar cases in the literature. He mentioned Müllers case of intraocular tuberculosis following a perforating injury as doubtlessly ectogenic.

Reference

Block, F. W., Klinische Monatsblatt für Augenh. 1921 Volume 67 Page 581.

DISCUSSION

Dr. A. B. Bruner, Cleveland, Ohio: Besides the clinical interest attached to most traumatic tuberculosis, many of these cases have an importance from the standpoint of industrial compensation, and it is possible to see that certain cases may develop medico-legal aspects. The number of cases reported in the literature is so few that I want to take the liberty of adding to the cases of Dr. Haessler one case which has come under our observation.

J. B., aged 29, white, was first seen July 25, 1929, with a history of having been injured by being struck in the left eye six days previously by an electric flash. He had been treated by his company physician who referred him to us. Examination showed the right eye entirely healthy; vision in the left eye 6/15. There was marked congestion and swelling just beyond the limbus and the cornea was slightly hazy; otherwise the media was clear. There was marked congestion of the conjunctival vessels, and the symptoms seemed to indicate impending necrosis and ulceration. During the whole course of the disease, however, neither necrosis nor ulceration has appeared. He was given the usual local treatment with bichloride ointment, hot packs, bandages and atropin. He showed little improvement and after a few weeks the eye began to get worse and he was sent to the hospital for further study. Complete physical examination was negative except for the presence of two abscessed teeth, and these were removed. Following the extraction of the teeth the condition did not improve, and a tuberculin test produced marked general, local and focal reaction. Before the tuberculin treatment was instituted neosalvarsan was tried, and intravenous therapy with typhoid vaccines, without success. Within ten days after the tuberculin treatment was started the condition began to improve. At present the congestion has practically disappeared and the vision is rapidly improving.

This case I think undoubtedly shows the inter-relationship of injury as an etiologic factor in post-traumatic ocular tuberculosis.

Dr. Edward Jackson, Denver: With reference to the infrequent reports of post-traumatic ocular tuberculosis, there are two things to be borne in mind. In Colorado, those of us who live there for any length of time come to recognize that tuberculosis is often present in the body but not diagnosed. This has been particularly enforced on some of

us in connection with ocular tuberculosis. But the general difficulty of excluding tuberculosis in any case is illustrated by the history of a young, apparently healthy dentist who had recently passed a perfect life insurance examination, who had hemoptysis from pulmonary hemorrhage. He was carefully examined physically by men who are expert in discovering pulmonary tuberculosis; they did not find anything. He had a second hemorrhage and was sent to the hospital. All the different methods of diagnosis were used, including tuberculin and careful x-ray studies of the lungs. The results were negative. They did not find any evidence of a lesion in either lung, that could be assumed to be the cause of this spitting of blood. A little later following a hemorrhage he developed pneumonia and died of the pneumonia. At autopsy they found, deep in the lung, a lesion that had run through the general course of tuberculosis. The total lesion was not larger than the end of the index finger. There was cavity formation, there were degenerating tuberculous deposits, caseation, all the changes of a tuberculous process at the periphery of the lesion—a lesion that was not discovered during life, and yet it was a lesion that killed the patient in a few weeks after the first manifestation.

In a case of tuberculosis of the macula, where early interference with vision directed attention to it, the patient had been through one attack before coming to Colorado and was sent there, not with a diagnosis of tuberculosis, but because she had relatives living there she was advised to give up her work as a stenographer for a time. I saw her in the second attack, and from the clinical appearance suspected tuberculosis. The use of tuberculin showed focal reaction in disturbance of the vision in the retina, and it also showed a bone lesion in the tarsal-metatarsal joint of the great toe. She had had some soreness there for two months; but, under the observation of a surgeon, tuberculosis in that position was not suspected until this reaction. The great toe was cured, she recovered from the choroidal lesion, and she had no symptom of pulmonary tuberculosis up to that time. But after that she had an attack, first of pleurisy and then lung involvement, that kept her in bed for four months. She finally recovered and is still active after several years in Denver.

This illustrates the difficulty of saying, even by the most careful examination, that there has not been tuberculosis present when an injury occurs. We have only learned within relatively few years the effect of trauma in producing outbreaks of parenchymatous interstitial keratitis. I feel sure that I have seen several cases with the first attack of the corneal lesion, from slight injury to the cornea. Formerly the first lesions recognized were bone lesions; that practically always were traced back to a history of injury, but a better understanding showed that the lesions were essentially tuberculous. We must recognize from these few cases the strong probability of a persistent and deep-rooted lesion, and that the cases are much more frequent than their appearance in the literature, or their diagnosis would indicate. We have under-estimated the importance of trauma in awakening tuberculosis, or providing a center for the tuberculous process, which must be recognized, for it is really very great.

Considering the whole subject together, and searching to discover if possible these cases of ocular tuberculosis, post traumatic, we shall find that cases of this sort are more numerous than we had supposed.

DR. A. N. LEMOINE, Kansas City: In my opinion Dr. Haessler has not definitely proven that his patient with uveitis following a cataract extraction, was tuberculosis. We have seen patients with identical clinical appearance, and course that proved to be due to lens cortex in a patient hypersensative to lens protein. Some of these patients will have deposits on the posterior surface of the cornea, and considerable pain that will continue for months, and eventually cause a blocked pupil. I feel that hypersensativity to lens protein should be eliminated in that case. However, I have seen patients with an old tubercular uveitis flare up, after trauma. Those who were in my conference, will recall that I mentioned a case of tubercular uveitis, and keratitis, that had been quiet for two years, that became active following a cataract extraction, and the eye was lost. This, however, was an exacerbation of an old tuberculosis, and not one precipitated in an otherwise normal uveal tract. I feel that all patients, having had a tubercular uveitis, should be immunized to tuberculin before any operative procedure is attempted as the artificial immunity last only a year or two and the operative trauma is very likely to precipitate a new attack.

DR. F. HERBERT HAESSLER, closing: I think the discussion has brought out the fact that the lesion is not as rare as a search of the literature would lead us to think. After all, the literature is an expression of what has been collected over a long period of time, yet several members have reported experiences indicating that much of the material that has been observed has not been deposited in the literature where it is available to the rest of us.

The difficulty in making the diagnosis, as emphasized by Dr. Jackson, is of course great, but that should not deter us from making the attempt. The commonest cause of chronic infection in this region are syphilis and tuberculosis, and it seems to me it is up to us to prove the absence of tuberculosis rather than to prove its presence.

As for the lens protein production of frank tuberculosis of the iris, I have never seen a case of that sort, and it has not occurred to us to rule it out.

OTOLARYNGOLOGIC SESSIONS

REPORT OF THE RESEARCH FELLOW IN OTOLARYNGOLOGY

W. J. McNally, M. D.
MONTREAL, CANADA

AN ANALYSIS OF THE LIMB RESPONSES TO SEMICIRCULAR CANAL STIMULATION IN THE FROG.

John Tait and W. J. McNally,

From the Departments of Physiology and Otolaryngology.
McGill University, Montreal.

It has been generally known for a long time that the labyrinth exerts some control over the movements and attitudes of an animal. As early as 1693 Bannister described balancers in certain flies. Flourens (1824) and De Cyon (1878) found that after injury or section of a semicircular canal in the pigeon, the rabbit and the frog, that the animal was very much disturbed and tended to fall over in the plane of the injured canal. (Byrne 1912). Kreidl found that interfering with the ear stone in the crayfish caused marked disturbance of equilibrium and by substituting iron filings for the otolith in the oto-cyst, the animal can be made to assume different attitudes by bringing a magnet near the head of the animal. The magnet causes a shifting of the iron filings in the oto-cyst. Kreidl also found that fish show a definite response to centrifugal force by leaning in toward the centre.

Two Groups of Labyrinthine Reflexes

Breuer (1874) was the first to point out two different groups of labyrinthine reflexes. He gave as his opinion that the reflexes due to movement arise in the semicircular canals and the reflexes resulting from position arise from the otolithic organs. For many years interest was centered chiefly around the semicircular canals and their effect upon eye and head movements.

Note: The authors wish to express their indebtedness to the American Academy of Ophthalmology and Oto-laryngology for the monetary grant made to W. J. McNally, Research Fellow of the Academy in Otolaryngology, part of which was used to defray some of the expenses of this research.

Otolithic Reflexes

Magnus and deKleijn were the first to make a detailed study of the labyrinthine reflexes with a view to determining more especially the otolithic reflexes and their effect upon the body musculature. They have shown that the otolithic reflexes are concerned with the movements and posture of the body as a whole and that they cooperate with and are influenced by reflexes from other parts of the body.

Semicircular Canal Reflexes

Our efforts have been devoted for the most part to the study of the control exerted by the semicircular canals upon the limbs and body musculature in contra distinction to the more widely known relation of the canals to head and eye movements.

Centrifugal Force

Our first attempt was to differentiate in a frog, subjected to turning about a vertical axis, between the response to pure angular acceleration and the response to centrifugal force. It is well known that if a frog is subjected to turning (angular acceleration) about a vertical axis in the horizontal plane, it responds by walking around in the opposite direction. If the frog is far away from the centre and the turning becomes rapid, the animal tends to lean in towards the centre. In our experiments we attempted to eliminate the effect of angular acceleration by increasing the speed of rotation so slowly as to render it imperceptible to the frog. In other words, angular acceleration was maintained below the threshold of stimulation for the frog. By this method, it was possible to place a frog on a turn-table, side to the centre, and to develop quite a high speed of rotation without the animal showing any tendency to make turning movements in any direction. We found, however, that as the speed of rotation was increased, the animal began to lean in towards the centre by flexing the limbs on the inside and extending the arm and leg away from the centre of rotation. This lean was increased until the speed of rotation became so high, the animal could no longer maintain his footing. A frog, after bilateral labyrinthectomy does not show this response to centrifugal force and like an inanimate object is thrown off the table as the speed of rotation increases. However, a frog with all six semi-

circular canals removed, though it does not respond to ordinary turning or angular acceleration, shows a normal response to centrifugal force when subjected to turning by the method just described. A frog with the nerves to both saccular maculæ cut also shows a normal response to centrifugal force.

This response of the limbs and body musculature to centrifugal force is labyrinthine in origin and most probably arises from the intact utricular maculæ. It is, therefore, an otolithic response, whereas the ordinary response usually described, which follows turning of an animal in the horizontal plane about a vertical axis is the true semicircular canal effect and is abolished after the removal of all six semicircular canals. Tait and McNally 1925.)

Gravity Stimulation

We also found that the posture or attitude assumed by a normal frog when subjected to centrifugal force could be elicited by simply tilting the frog slowly out of the horizontal plane. It responds by flexing the upper arm and leg and by extending laterally the arm and leg on the lower side, the result being to maintain the head in, as nearly as possible, the horizontal plane. This response is not possible in a frog with both labyrinths removed. It is possible to elicit this response after removal of the ampullæ of all six semicircular canals and after section of the nerves to both saccular maculæ. This protective response, therefore, also arises from the utricular maculæ. In this last case, the attitude is assumed in response to gravity stimulation. The head is maintained at right angles to the pull of gravity whereas when the frog is subjected to centrifugal force, he adopts an attitude wherein the head is maintained at right angles to the resultant of the pull of gravity and the pull exerted by centrifugal force at any given moment during the turning. This is an otolithic reflex—utricular in origin.

Stimulation of Horizontal Semicircular Canal

As we have already mentioned an animal subjected to rapid turning in a horizontal plane responds by turning its head and eyes in the opposite direction and in some cases as in the frog, the animal tends to walk around in the opposite direction, as though it were trying to retain its original field of vision. Ewald

found that when one horizontal canal was injured in the pigeon, the animal responded normally to turning to the intact side, whereas there was only a very slight response of the head and eyes to turning to the operated side. Ruttin found that this is also the case in the human, that there is a slight response to turning to the operated or diseased side. Maxwell, McNally and Tait and others found that after destruction of one horizontal ampulla in the frog that the animal responds normally to turning to the intact side, but there is no response whatsoever to turning to the operated side.

Stimulation of a Vertical Semicircular Canal

De Cyon (1878) found that after injury to the vertical canals in the frog, the animal was very much disturbed and that it tended to fall in the plane of the injured canal. In our investigation of the semicircular canals in the frog, rather than try to injure the canal, we attempted to completely destroy by cautery, the ampulla of a particular canal without injuring any of the other labyrinthe structures. After destruction of a vertical ampulla, we found that when at rest the animal was for all practical purposes quite normal. However, if the animal was tilted out of the horizontal plane about a horizontal axis, it lacked some of the protective movements which are executed so promptly by the normal frog.

The normal frog when quickly tilted out of the horizontal plane, makes a rapid protective movement or readjustment of the limbs, which tends to maintain the head in its original position in space. For example, if a frog is quickly tilted forward, the head is raised off the table, the arms being extended forward. If the animal is tilted backwards quickly, the head is lowered, the arms are extended laterally and slightly backwards and the legs are extended backwards. This rapid readjustment of the limbs and of the body places the animal in the same attitude or posture as that which is assumed when the animal is very slowly tilted out of the horizontal plane as has been previously described. This rapid response to tilting out of the horizontal plane is not present in a frog after a bi-lateral labyrinthectomy nor is it present after removal of all six semi-circular canals. This rapid protective movement is, therefore, elicited from the intact semicircular canals.

Earlier Impressions

In our earlier attempts at analyzing the protective reactions of a normal frog to rapid tilting about a horizontal axis, we came to the conclusion that a semicircular canal responded best to a movement in its own plane and that it responded to tilting in only one direction, and that a vertical canal controlled chiefly the movements of the limbs of the corresponding corner of the body. If a normal frog is tilted forward in the plane of one of the anterior vertical canals, the most obvious response is a thrusting or extension forward of the arm of the corresponding corner of the body. If the tilting is in the plane of the right anterior vertical canal, the right arm is extended forward, the right corner of the head being raised. If the ampulla of the right anterior vertical canal is destroyed and the animal is tilted forward in the plane of this canal, the head dips forward and to the right, the most evident abnormality being a failure to extend the right arm. Hence the conclusion that a vertical canal controls chiefly the movement of the limb of the corresponding corner of the body. In spite of this conclusion it seemed obvious that following the destruction of the ampulla of any one vertical canal, there was more than the lack of extension of the limb of the corresponding corner of the body—a lack of some movement in the other three limbs—but as these things were not so obvious and were more difficult to describe they tended to be overlooked. It seemed probable that one semicircular canal was concerned with the rapid response of the limbs of both sides of the body and subsequent observation seems to confirm this.

Reaction of a Normal Frog

When a normal frog is placed on a tilt-table having a horizontal axis and the frog is rapidly tilted to either side, it makes a very rapid readjustment of the limbs which tends to maintain the head in the horizontal plane. There is no swaying of the head or body. If the tilting is to the left, the left arm is extended laterally, the right arm is flexed and is held close to the body. This readjustment is so rapid that the head does not seem to move from its original position in space. If the tilting is through a small angle in space, the palms of the hands do not shift their base, but if the tilting is through a wide angle, the

left arm as well as being extended, moves farther down the board, and the left leg may be extended laterally.

If the frog is rapidly tilted backwards through a small angle, the arms are flexed so that the head is brought nearer to the table and the legs may be slightly extended, that is the knees tend to move laterally from the side of the body. However, if the tilting backwards is through a wide angle, the arms extend laterally, the palms of the hands being turned backwards as though prepared to make a "breast stroke" as does a man when swimming. The legs are extended backwards.

When rapidly tilted forwards through a small angle, the arms are extended forward so that the head is raised from the table. If through a wide angle, the arms are moved forward on the board. The legs do not seem to move, but the body seems to move back on the flexed thighs.

These protective reactions to rapid tilting are evidently complicated movements and they involve the limbs on both sides of the body—all four limbs in each movement. If both labyrinths have been removed or if the ampullæ of all six semicircular canals have been destroyed (McNally and Tait, 1925) these protective movements to rapid tilting are abolished. When quickly tilted out of the horizontal plane in any direction, such an animal simply falls over as the rapid protective movements do not occur. If the utricles are still intact, that is if only the canals have been removed, the animal later picks itself up and slowly assumes the normal protective attitude for the new position. This is a utricular response to gravity and not the rapid protective response from the semicircular canals. These rapid protective reactions are, therefore, dependent upon an intact semicircular canal mechanism.

ABLATION EXPERIMENTS UPON THE SEMICIRCULAR CANALS

The proof that one semicircular canal exerts an influence over the movements of the limbs of the opposite side of the body will be seen from the following experiments:

In all the experiments about to be described where one or more of the ampullæ of the semicircular canals have been injured, the reactions of the animals to centrifugal force and to slow tilting or gravity reactions are all normal and, therefore, will not be referred to again.

Destruction of the Ampulla of Left Horizontal Canal

When placed on a turn-table and turned to the right, a frog reacts normally by walking around to the left. This requires the normal movements of all four limbs. When the animal is turned to the left, it does not react but remains quietly at rest. The intact right horizontal semi-circular canal has evidently elicited reactive movements from all four limbs during turning to the right.

Destruction of the Ampulla of the Left Anterior Vertical Canal.

When placed on a tilt-table and tilted rapidly to the right, the animal responds normally by extending the right arm and flexing the left, so that the head is held in the horizontal plane without any swaying. When tilted to the left, the head sways slightly to the left, the left arm not being promptly extended nor the right arm flexed. This absence of flexion of the right arm is quite as definite as the absence of extension of the left arm. The rapid tilting to the left, therefore, throws the head slightly to the left before the flexion of the right arm and extension of the left arm takes place. In our earlier description (McNally and Tait, 1925) of this experiment, we failed to stress the fact of the absence of the flexion of the right arm. When tilted to the right, the intact right anterior vertical semicircular canal elicits the normal extension of the right arm and flexion of the left arm, therefore, influencing the limb movement on both sides of the body. If the animal is tilted forward in the plane of the injured left anterior vertical canal, the abnormal response is more marked, the head tilts downward and to the left, the left arm not being promptly extended forward and laterally; the right arm is not flexed and rather gives the impression of some extension. There is no evident movement of the hind limbs. It is only after an appreciable time that the animal picks itself up and assumes the normal protective attitude for the new position. When tilted backwards in the plane of the injured canal, the response is normal. When tilted forward, the head dips down and to the left, the left arm not being promptly extended. The right arm is extended and seems to push the head over to the left. When tilted backwards, there is no obvious abnormal response.

Destruction of the Ampulla of the Left Posterior Vertical Canal.

If tilted backward in the plane of the injured canal, the head tends to go backwards and to the left. It is raised off the table evidently due to some abnormal response of the right arm, which is difficult to make out. It may be the absence of the "breast stroke" with the right arm, which would tend to pull that corner of the body downwards and forwards. The legs are not extended and if the tilting is rapid and through a wide angle, the animal is very easily thrown off the table. If tilted forward in the plane of the injured canal, the reaction is normal. When tilted strongly backward, that is the long axis of the frog being at right angles to the axis of the table, the head sways back and to the left. The right leg is extended back and to the right, the left leg remains flexed or may be extended just a little. The arms seem to extend laterally but evidently not normally because the right side of the head is raised. When tilted forward the reaction is normal. When tilted back in the plane of the intact right posterior vertical canal, the response is normal, that is the head is lowered to the table, both arms are extended laterally, the palms facing backwards, and both legs being fully extended. This normal extension of the arms and the extension of both hind legs when tilted in the plane of the intact posterior vertical canal is evidence of the effect that the intact posterior vertical canal elicits normal cooperative movements from all four limbs.

Destruction of the Ampulla of Both Posterior Vertical Canals.

When the animal is placed with its long axis at right angles to the axis of the tilt-table and tilted forward, the reaction is normal. When tilted backward, the head raises off the table backwards, and there is an absence of lateral extension of both arms—the "breast stroke" previously described. The legs are not extended and if the tilting is through a wide angle, the animal is very easily tumbled over backwards. In this experiment the absence of the posterior vertical canals is responsible for an abnormal movement of both the arms and the legs.

Destruction of the Ampulla of Both Anterior Vertical Canals.

When the frog is placed on the tilt-table with its long axis at right angles to the axis of the table and is tilted backward,

the reaction is normal. When tilted forward, the head slews downwards and forwards, the arms instead of being promptly extended forward seem to be thrown out laterally and though there is no obvious movement of the legs, the body seems to slide away from the flexed thighs. After an appreciable time, the animal picks itself up and assumes a normal protective attitude. This experiment shows control of the anterior pair of canals over the fore and hind limbs.

Destruction of the Ampulla of All Three Semicircular Canals of the Left Side.

When tilted forward the response is that of an animal with a destroyed left anterior vertical canal. When tilted backward, it shows the response of an animal after destruction of the left posterior vertical canal. When tilted to the left, it shows a slightly more exaggerated response of an animal with the left anterior vertical canal destroyed. When tilted to the right, the response is normal. On the turn-table, there is no response to turning to the left.

Destruction of the Ampulla of All Six Semicircular Canals

To rapid tilting in any direction the animal slews over in that direction making no prompt effort to protect itself. It has lost its rapid protective reaction to movement in any direction.

SUMMARY

From these experiments, it seems evident that a semicircular canal rather than affecting the movements of any one limb or of any one group of muscles is directly concerned with the protective reaction of all four limbs to movement in its own plane, that is, it controls a different group of muscles in different limbs depending upon the direction and plane of the movement. It would seem more correct to speak of a semicircular canal as controlling all groups of muscles concerned in the rapid protective reaction to a sudden movement in its own plane, in the direction in which the ampulla points.

EXPERIMENTS ON THE RECORDING TILT-TABLE

In attempting to make a more careful analysis of these various limb responses to semicircular canal stimulation, the idea was conceived by Dr. Tait to separate a decerebrate frog into

two components. This was accomplished by dissecting free the sciatic nerve, the thigh bone and muscles being cut away, leaving the lower leg segment connected to the body component by the intact sciatic nerve only. In this way it proved possible to subject the body component to rapid tilting while the limb which rested on an adjoining table is only affected by impulses carried to it by the intact sciatic nerve. It was found that in the decerebrate frog otherwise normal, backward rapid tilting of the body component caused a kick of the limb component.

Recording Tilt-Table

An apparatus was gradually evolved whereon the flexor and extensor muscle groups of the ankle could be connected each to a lever recording on a smoked drum. The apparatus became further modified till in its present form it is possible to investigate the extensor and flexor muscle groups of both ankles simultane-

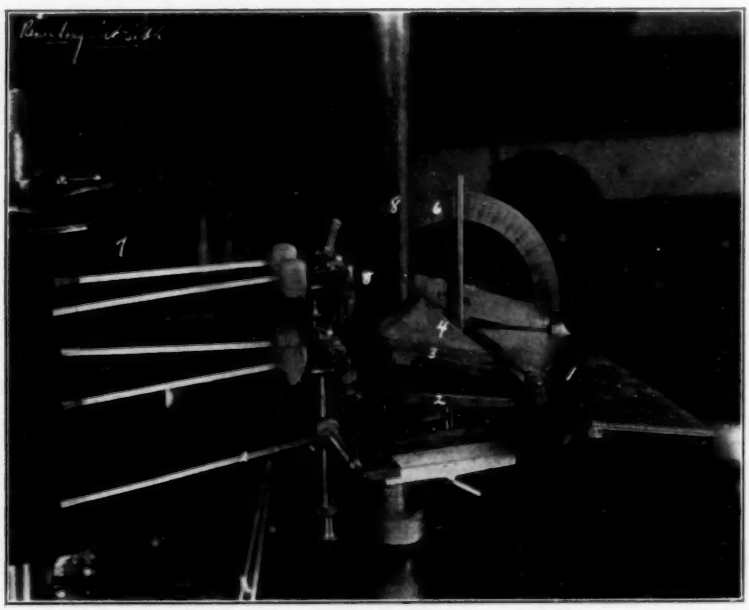

Figure 1—THE RECORDING TILT-TABLE

1. Tilt-table.
2. Recording lever from tilt-table.
3 and 4. Fixed Table.
5. Joint for swinging away the two top levers while attaching the threads to the lower levers.
6. Protractor for measuring angle of tilting.
7. Drum.
8. Supporting Rod.

ously. The apparatus consists of a fixed stand to one side of which is attached a small table which may be tilted up or down. To the other side of the fixed table are arranged four levers in pairs, one above the other, the points of which are so arranged as to write on a vertical drum one above the other. (See Figure 1). From the tilt-table there is also a lever which records the movements of the tilt-table, thus from the kymogram can be seen the exact moment the tilt begins and the rapidity with which it is carried out, and the height of the tilt, etc. A time tracing is added from an electrically controlled tuning fork. From a study of the kymogram, therefore, it becomes possible to calculate the length of time taken by a muscle group to respond from the moment that the tilt is given to the body segment.

Preparation of the Animal

Both limbs are prepared by dissecting out the sciatic nerves and removing the thigh portion of the leg. The tendons of the

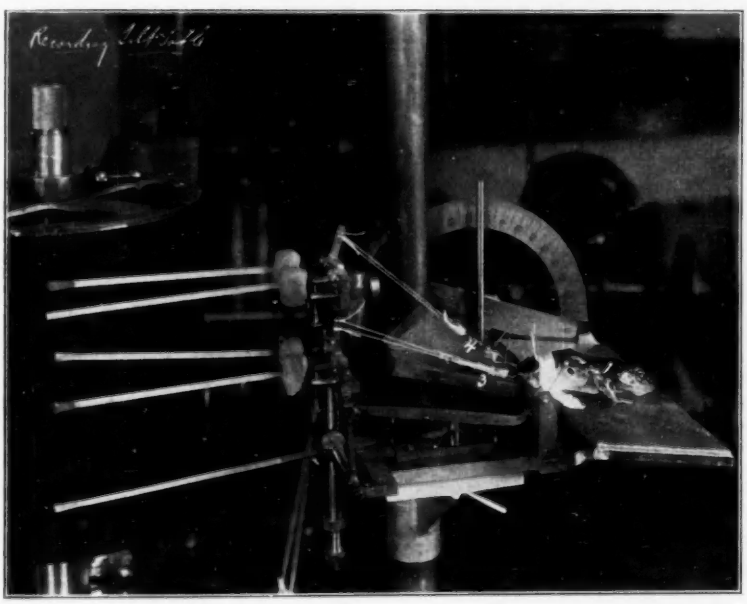

Figure 2

1. Decerebrate frog in position on the tilt table.
2. Restraining band. (Note the two sciatic nerves bridging the axis and running to the two lower leg segments).
3 and 4. Right and left legs with threads from the flexor and extensor muscle groups of the ankles to the recording levers.

anterior or flexor group and posterior or extensor muscle group of the ankle joint are then dissected out and a ligature applied to each. The foot is then amputated at the ankle. The leg segment is held steady to the fixed table by transfixing the knee joint with a pin. The ligatures from each tendon group are then attached each to a separate lever. The body segment is fixed to the adjoining tilt-table by cotton bands. The sciatic nerve loosely bridging the axis of the table. (See Figure 2). The nerve is kept moist by continuous saline applications. The body segment may be arranged with the long axis of the frog at right angles to the axis of the tilt-table or it may be rotated to either side in the plane of one of the posterior vertical canals.

Preoperative Examination

Before any operation is performed, the animal is carefully examined on the turn-table and on the ordinary tilt-table for the reactions of the normal frog to rapid turning about a vertical axis and to slow and fast tilting about a horizontal axis. Using the new recording tilt-table, the reactions of the normal decerebrate frog were first studied, then different labyrinthine operations were performed and the resulting effect upon the recording muscle groups were studied from the kymograms.

Some Observations Derived from Examination of the Normal Frog

The most striking thing under the conditions of these experiments is that the animal does not respond to mere slow tilting or gravity stimulation, i. e., the muscle groups connected to the levers show no response to the slow moving up or down of the tilt-table.

Repeated examination of the normal decerebrate frog on the recording tilt-table revealed the fact that to backward tilting the flexor muscles of the ankle of each side show more constant and greater response than the extensor muscles—the gastrocnemii.

The character of the response of the muscle groups of the ankle joint to semicircular canal stimulation is "twitch-like" and reminds one of a tendon-jerk. They can be readily and regularly graded by the degree of angular acceleration imparted to the head body component on the tilt table. From the tracing or

kymogram, it is possible to measure the time required from the beginning of the tilt to the occurrence of the muscle twitch, which is the latent time of the reflex muscle response. By measuring this off on the time tracing, the actual reaction time is arrived at. The shortest recorded reaction time of the muscle group response to semicircular canal stimulation is 35σ.

EXAMINATION AFTER OPERATION

Bilateral Labyrinthectomy.

When such an animal is examined on the recording tilt-table, there is no response from any of the recording muscle groups no matter how rapid the tilt nor what the orientation of the body segment. However, if the table is jarred, there is an immediate response of all four muscle groups (stimulation of receptors other than the labyrinths).

Destruction of the Ampulla of All Six Semicircular Canals.

When on the recording tilt-table there is no response from any of the recording muscle groups to tilting of the body segment, no matter how rapid or in what orientation. From these two experiments, it becomes evident that the "twitch-like" response of the muscle groups of the ankle to backward tilting of the body segment is labyrinthine in origin from the semicircular canal mechanism, as it is abolished by their removal. It is not otolithic in origin.

Right Sided Labyrinthectomy.

When the flexor and extensor muscle groups of both ankles are recording and the body segment is arranged with its long axis at right angles to the axis of the table, and the table is tilted rapidly backwards, the greatest and most constant response is from the flexor muscle group of the left ankle. The response from the extensor muscle group of the left ankle being less marked. There is only an occasionally weak response from the flexor muscle group of the right ankle and even less marked from the right extensor muscle group.

In the unilateral delabyrinthized preparation, a fairly constant reflex of the arms was noted. Owing to the unilateral labyrinthectomy the animal leans down to the operated side, that is after a right sided labyrinthectomy, the right arm is flexed and

the left arm extended. The body segment was restrained by a cotton band over the pelvis. To backward tilting the right arm is moved outwards, backward and forwards like the movement of a man swimming breast stroke when he wishes to dive forwards and downwards. On completion of the upward tilt, the arm is flexed and returned to its original position. The left arm is slightly more extended, the palm being turned backwards and upwards. By these movements both arms are often raised off the ground so that the animal rests on its sternum. This reflex seems most marked in these unilateral delabyrinthized animals. It seems to be an exaggeration of the normal lateral extension movement of the arms in response to extreme backward tilting.

Destruction of the Ampulla of the Three Semicircular Canals of the Left Side.

Examination before preparation of the limbs for the recording table showed the gravity reactions to be normal, that is both utricular maculæ were responding. The animal showed the usual abnormal responses to tilting in the planes of the absent canals. When on the recording tilt-table, to backward tilting, the arms showed the "breast stroke" reflex previously described but less marked than in the animals with unilateral labyrinthectomy. The chief muscle response to backward tilting was from the flexors of the right ankle. There was a less marked response from the flexors of the left ankle. The extensors of both ankles scarcely responded at all.

Destruction of the Left Posterior Vertical Ampulla.

This experiment will be described in more detail to give a better idea of the method of procedure.

May 13th, 11 A. M. Under ether anæsthetic, using a previously examined decerebrate frog, the left posterior vertical ampulla was exposed through the roof of the mouth and the ampulla was destroyed with the actual cautery.

4 P. M. Frog in good condition. On ordinary tilt-table shows loss of response to tilting in the plane of the left posterior vertical canal. The hind limbs were prepared for the recording tilt-table by dissecting out the sciatic nerves and severing the thigh. Only the flexor muscle group of each ankle was attached to the recording levers.

Tracing 1

Long axis of the body at right angles to axis of tilt-table. The only muscle response was from the right flexors. (See Fig. 3).

Tracing 2

The same orientation of body segment. Chief response is from the right flexors, there being a slight response from the left flexor muscle group.

Tracing 3

The body segment slewed over to the left in the plane of the injured left posterior vertical canal. To backward tilting, there is no response from either recording muscle groups. (See Figure 4).

Tracing 4

Frog slewed well to the right in the plane of the intact right posterior vertical canal. Backward tilting elicited a very marked response from the right flexors and a less marked response from the left flexors. (See Figure 5).

Tracing 5

Same orientation of the body segment. Backward tilting elicited a marked response from the right flexors and a less response from the left flexors.

Tracing 6

Animal slewed well to the left. No response from either muscle group except when table was jarred when both muscle groups responded.

Tracing 7

Same orientation of body segment. To backward tilting no response from either muscle group.

Tracing 8

Body segment slewed to the right in plane of the right posterior vertical canal. Backward tilting elicits a marked response from the right flexors, and a less marked response from the left flexor muscles of the ankle joint.

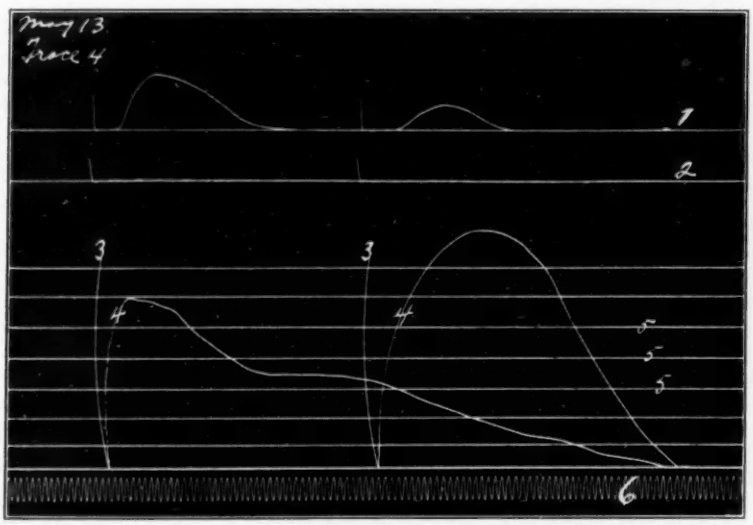

Figure 3

Tracing from frog with the ampulla of the left posterior vertical semicircular canal destroyed. Response to backward tilting when the long axis of the frog's body is at right angles to the axis of the table.

1. Flexor muscles of right ankle.
2. Flexor muscles of left ankle.
3. Record of tilt-table when drum at rest.
4. Record of actual tilt of table during experiment.
5. Each line denotes 10 degrees of tilting.
6. Time tracing. (Note the marked response from the right flexor muscles.)

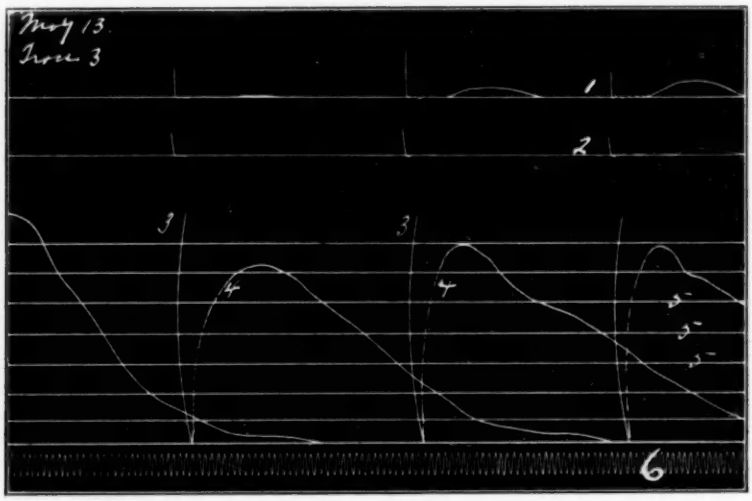

Figure 4

Backward tilting with body component slewed to left so that the tilting is in the plane of the destroyed left posterior vertical semicircular canal. (Significance of numbering as in Fig. 3). Note the absent or only very weak response to tilting so extreme as to jar the apparatus.

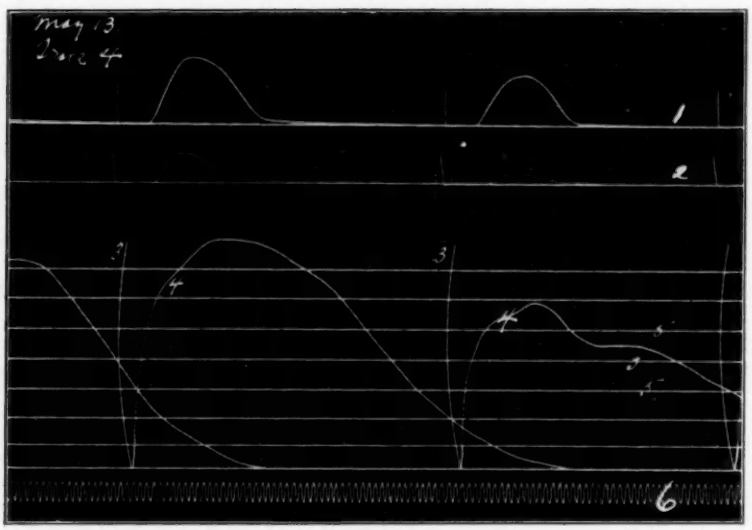

Figure 5

Frog slewed to the right so that the backward tilting is in the plane of the intact right posterior vertical canal (the significance of the numbering is the same as in Fig. 3). Note the very marked response of the flexors of both limbs though chiefly of the right flexors to relatively slow tilting.

This experiment furnishes very definite evidence to the effect that one posterior vertical canal may elicit a response from the limbs on the opposite side of the body.

Destruction of the Ampulla of Both Posterior Vertical Canals.

Only one such animal was tested on the recording tilt-table, so that one cannot conclude very much from the result, but it is interesting to note that with all four muscle groups recording to backward tilting, there was no response from any of the muscle groups, no matter what the orientation of the body segment. This frog was in good condition.

Destruction of an Anterior Vertical Ampulla.

Injury to one or both anterior vertical canals did not have any definite or constant effect upon any of the four recording muscle groups of the ankle joint when the frog was subjected to backward tilting.

Post Mortem Examination

The experiments described were all controlled by carefully performed post mortem examinations of the labyrinthine lesion using the dissecting microscope. It was hoped to have microscopic serial sections of some of these labyrinths, but as they are not yet ready for section, they cannot be included in this present pre-sessional copy.

Summary

This method of examining the leg muscle response on the recording tilt-table has added a very striking proof to what we already observed, that is that the limb responses to semicircular canal stimulation are an extremely rapid and prompt protective response to any sudden movement or acceleration affecting the head body component of the animal. The reaction time of the recorded twitch being only 35σ. The "twitch-like" character of the response shows that it is not maintained, but as we have already pointed out the new position which is so rapidly assumed must be maintained by reflexes from the more slowly responding utricle, which are static in character.

OTHER REFLEXES WHICH INTERFERE OR COOPERATE WITH THE LABYRINTHINE REFLEXES

The absence of response to gravity stimulation under the conditions of the experiments on the recording tilt table was at first surprising. To hold the head and body component and to prevent pulling on the sciatic nerve, the head body component is fixed securely by cotton bands which interlace over the back of the frog and are pinned to the cork of the tilt-table.

As a control of this retaining method to see if it might explain the absence of gravity responses, several experiments were carried out.

A simple instrument was devised consisting of a small piece of cork which just covers the under surface of the frog's abdomen. The skin of the abdomen is pinned to the edge of the cork on each side, fore and hind, in such a way that the limbs are quite free to move. To the bottom of this cork a rod may be attached so that the animal can thus be raised off the ground.

Examination of the Normal Frog

If a decerebrate frog which has been previously found to respond normally to slow and fast tilting is attached to the cork platform, it still jumps about and responds normally to slow and fast tilting, even with the cork attached to its abdomen. However, if the platform is raised off the ground and is held by the rod so that the limbs are now free in the air, the frog no longer responds to slow or fast tilting. It seems to have lost its gravity responses and does not seem to show any of the head righting responses which are described by Magnus in mammals. In some cases, if a platform is slid up the rod to make a solid contact for the animal's legs, the frog may assume a normal attitude for whatever position it is in, but as a rule there is very little response to either slow or fast tilting. If this same frog is now freed from the platform, it immediately responds normally in every way to slow and fast tilting and jumps about normally.

The absence of limb contact with the ground or the restraining of the body segment to the platform has in some way interfered with the normal labyrinthine responses of the animal, so that the restraining of the body segment under the conditions of

our experiments on the recording tilt-table may explain the absence of response to slow tilting or gravity stimulation.

Another experiment was tried which consisted of applying a mattress suture to the skin of each side of the abdomen. One suture was applied behind the insertion of the arms and another just in front of the insertion of the thighs. The sutures were tied tightly, thus making two tight bands of skin about the animal's body. These frogs responded normally to slow and fast tilting. In other animals tight ligatures were tied around the body behind the arms and around the pelvis. These animals also responded normally to slow and fast tilting. It is, therefore, not mere tight pressure on the body segment which interferes with the labyrinthine responses.

Examination of the Operated Animal

Several experiments were carried out on unilateral delabyrinthized frogs. When pinned to the platform and held with the limbs free in the air, the body is held almost normally symmetrical. The lean to the operated side almost completely disappears, the arms being flexed under the platform in a clasp reflex, both arms being quite symmetrical. If a flat surface is slid up under the animal to give its arms and legs contact, immediately the limbs on the operated side are flexed and the limbs on the intact side are extended. These experiments would seem to show that a tactile or kinæsthetic factor from the pads of the feet is also necessary for the maintenance of the characteristic unilateral labyrinthectomy posture.

It was also observed that when a frog with unilateral labyrinthectomy was carefully handled and the twist of the body was undone by clasping the head between the thumb and forefinger and slowly bringing the head into the horizontal plane, the new position of the head is maintained so long as the animal is undisturbed. Breathing movements are not sufficient to undo the new posture. Jarring of the table, etc., however, immediately brings about a reassumption of the usual unilateral labyrinthectomy posture.

Summary

From these control experiments, it becomes evident that as shown by Magnus for the mammal, there are also in the frog

other reflexes which supplement and modify the labyrinthine reflexes in controlling the posture and movement of the normal frog, even though the labyrinthine righting reflexes on the head are not so evident in the frog as they are in some higher animals. It is, therefore, not surprising that in our recording tilt-table experiments where the hind limb are severed and where the body segment of the frog is bound to the table, there is no evident response of the muscle groups of the ankle joint to slow tilting or gravity stimulation.

SUMMARY

In earlier experiments we were able to show that a frog subjected to turning about a vertical axis makes a different response to centrifugal force than it does to angular acceleration. The response to angular acceleration is the generally known turning in the opposite direction to the turning of the table. This is abolished by removal of all six semicircular canals. The response to centrifugal force is a leaning towards the axis of turning. This reaction is absent after bilateral labyrinthectomy but it is not affected by removal of the ampullæ of all six semicircular canals or by section of the nerves to both sacullar maculæ. This reaction to centrifugal force, therefore, most probably arises from the utricular maculæ.

The same attitude which is assumed in response to centrifugal force can be elicited by simply tilting the frog out of the horizontal plane. It responds by leaning up the tilted plane as it were. This latter response is a reaction to gravity stimulation.

After removal of all six semicircular canals, it was found that the frog still responds to gravity and centrifugal force, but that it has lost its prompt protective response to rapid movement or tilting. Injury or destruction of the ampulla of a semi-circular canal causes a loss of the prompt protective response to any sudden movement in the plane of the injured canal. It was first thought that the absence of prompt extension of the limb of the corresponding corner of the body was the chief result of injury to a single vertical canal. More careful investigation has since revealed the fact that the protective response to tilting in any one direction consists of extension of the limb on the side toward

which the animal is tilted and flexion of the limbs of the opposite side. After destruction of one ampulla, the absence of flexion in the limbs of one side is just as marked as the absence of extension of the limbs on the side to which the animal is tilted. It, therefore, seems more correct to consider a semicircular canal as influencing the response of all four limbs to any sudden movement in the plane of that canal, and in the direction in which the ampulla points.

This has been further substantiated by examining the animal in two components on an instrument specially designed—a recording tilt-table—whereon the muscle groups of the ankle joint are connected to levers so that the responses may be graphically recorded.

From these experiments it was determined that the response to backward tilting of a decerebrate frog otherwise normal is a "twitch-like" response, the reaction time of which is exceedingly short, 35σ, confirming the idea of the rapidity of the protective response from the semicircular canals. The flexor muscles of both ankles respond to backward tilting more than the extensors and after unilateral labyrinthectomy or after destruction of the ampulla of one posterior vertical canal, the response is more from the flexor muscles of the ankle of the intact side.

Tilting in the plane of the injured canal elicits very little response from any muscle group, whereas tilting in the plane of the intact posterior vertical canal elicits a fairly marked response from both hind limbs and also from the fore limbs showing that the intact canal is influencing the movement of all four limbs.

It was found that the head righting reactions described by Magnus in the mammal are not so evident in the frog. Strapping the animal or removing contact from the feet does interfere with its usual labyrinthine reactions showing that as described by Magnus for the mammal, there are other reflexes which supplement or modify the labyrinthine reflexes controlling the posture and movement of the normal frog.

Conclusions

1. Reactions of a frog to turning about a vertical axis are in response to two factors—acceleration and centrifugal force.

2. The tendency to turn in the opposite direction to the rotation is in response to angular acceleration and is elicited from the semicircular canals.

3. The tendency to lean in to the centre of the turning table is in response to centrifugal force and is most probably from the utricles.

4. One vertical semicircular canal influences the protective responses of all four limbs to rapid tilting in the plane of that canal, and in the direction in which the ampulla points.

5. One posterior vertical semicircular canal elicits a response from the flexor muscles of both ankles, but more marked from the muscles of the same side.

6. The muscle response to semicircular canal stimulation is "twitch-like" and extremely rapid, the shortest recorded reaction time being only 35σ. The frog does not show the labyrinthine head righting reactions, which Magnus described in the mammal.

7. The labyrinthine reflexes in the frog are affected by the reflexes from other parts of the body, which supplement or modify them in controlling the posture and movements of the normal frog.

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This paper was not open for discussion.—*Editor.*

THE RELATION OF THE EAR, NOSE AND THROAT TO GENERAL INFECTIOUS DISEASE.

JOHN F. BARNHILL, M. D.

INDIANAPOLIS, IND.

That primary focal infection is responsible for all secondary diseases of an infectious nature is now conceded by most bacteriologists, pathologists, internists and otolaryngologists. It is also generally recognized that of all primary foci of the body responsible for secondary general infection, those located in the mouth, nose, throat and ear stand first in relation to the causation and severity of secondary general disease.

The list of bodily ailments that have been traced to an origin in some focus in the head or elsewhere is long and includes a large percentage of the most distressing, most serious human afflictions. Chief among these are the inflammatory affections of the heart, kidneys, gall bladder, skin, appendix, joints, muscles, lymph nodes, nerves, meninges, stomach, intestinal tract and eyes.

The severity of these affections varies from ailments which are but transitory and free from danger to diseases characterized by great chronicity, prolonged suffering, much deformity, and serious or even fatal involvement of vital organs.

Mortality statistics show that death occurs oftener from disease of the heart than from any other affection, and leading internists believe that a vast majority of such fatalities from heart lesions are caused primarily by foci of infection situated chiefly in the head. Professor Davis, of the University of Illinois, emphasized this belief in the statement that there are in the United States fully 2,500,000 persons who suffer from heart disease which had its origin chiefly in streptococcic tonsillar infection. This belief in the positive relationship between primary foci and secondary general disease, as well as in the frequency of occurrence of the secondary infections, is expressed by Professor John Kolmer of the University of Pennsylvania when he says that of the total number of deaths recorded, eighty to ninety per cent are due to infection, and that

such infection originates primarily in some local focus. Esterman makes an equally broad statement concerning the deleterious effects of focal infection when he states it as his belief that a majority of all sick people suffer in some way or other from disorders traceable to such foci. And Crowe still further strengthens this belief in the assertion that thousands of people suffer from some general disability as an end result of focal infection.

During the past three decades many articles have appeared, sponsored by clinicians whose experience and candor command utmost respect, all tending to show a focal origin of infection for an almost incredible number of bodily ailments that formerly were thought due to other causes. A review of these opinions and a study of clinical records must convince the student of medicine that the question of focal infection will remain one of the great problem in medicine and surgery, and that the development of procedures looking toward prevention of such foci should continue to excite paramount interest among medical men. My own convictions, based on an uninterrupted experience, both in teaching and private hospitals, that covers the whole period during which the subject has maintained leading interest, are in full accord with those who believe in the focal origin of general disease, and that a great majority of primary foci are located in the head. During this more than a quarter century the conviction has steadily grown upon me that should it ever be possible to prevent the establishment of primary seats of infection in the head or to eradicate them completely before secondary involvement takes place, a large percentage of the most troublesome and dangerous human afflictions would be completely banished from the earth.

The chief problems connected with prevention and eradication of such primary foci fall wholly in the field of the otolaryngologist.

Many facts of sufficient interest have been established concerning the mode of primary invasion of the body by pathogenic bacteria, and the reaction of the tissues to the invaders to justify a brief account of a few that are most nearly related to the question under discussion. Some of these are sufficiently trite, but for purposes of emphasis will be restated.

The tissues and fluids of the body contained within the skin and mucous membranes are sterile at birth and will remain sterile through life unless contaminated from outside sources.

Bacteria of several varieties and of different degrees of pathogenicity inhabit the surface of both the mucosa and integument at all periods of life, but their presence in these situations does not constitute infection, for it is only when bacteria in sufficient numbers and of great enough virulency actually penetrate these protecting barriers, and gain lodgment in the tissues within that infection is possible. The processes incident to the passage of pathogenic organisms through the coverings of the body, their establishment of residence there, and the resistance of invaded tissues to their entrance and continued presence has been aptly compared to the actions of opposing, hostile armies in active warfare.

In the warfare between bacteria and bodily tissues, bacteria are always the aggressors, whereas the bodily structures are always on the defensive. Each side possesses its own peculiar advantages and weaknesses. Sometimes the bacteria, sometimes the tissues are victorious. Failure of the bacteria to survive, and therefore failure to establish a focus of infection after entering the tissues may depend either on the small number of organisms engaged, or upon their lack of virulence. Failure may also be due to a too powerful resistance developed on the part of the invaded tissues to the presence of the invaders.

Bacteria are not capable of penetrating the protecting, surface epithelium of either the skin or mucous membrane so long as it remains normal. While it is true that Van Klecki and Wood have demonstrated the possibility of some types of bacteria passing through uninjured or unbroken epithelial surfaces, the phenomenon has been explained on the ground that whereas the surface cells seemed anatomically perfect, they were in fact physiologically weakened and therefore abnormal. It may be said therefore, that it is only in the event of an actual anatomical break or physiological weakening of the surface area that it is possible for local infection to take place, or secondary general involvement ever to occur.

The injury which opens a pathway to the entrance of infecting germs may be violent, in which event the wound may be large and visible; or the traumatism may be of trivial, insignificant nature and the resulting wound be correspondingly slight and wholly invisible. Equally severe and dangerous secondary infections may enter the body through a gaping, extensive wound, or through an invisible and therefore often unsuspected injury

Injury to the mucosa of the upper air tract is almost wholly of the latter type. Visible evidence of violent traumatism in that region is rare, and easily overlooked.

While, as stated, infection may take place through surface injury on any part of the body, it is common observation that certain areas, chiefly in the head, are predisposed to focal infection, for the reason that they are so unfavorably situated as to be subjected to constant irritation from inhalation of dust or other impurities, to frequent atmospheric changes, to lodgment and growth of bacteria and to retention of secretion.

Should the organisms succeed in passing the broken barrier of surface epithelium and in entering the sterile tissue within, they must still meet and overcome a second barrier before it is possible for them to establish a focus of infection in the invaded structure. This defense consists of the action of the chemical and morphological elements of the tissues and blood. Stimulated by the presence of the bacteria, additional quantities of antitoxin are liberated. Increased lymphocytosis and leukocytosis takes place. The invaders are crippled or killed by the antitoxin, and are attacked, enveloped and finally destroyed in so far as possible by the phagocytes.

Should the invading organisms be overcome by the foregoing defense mechanism the invasion will come to an end before a focus of infection can be established. Should it continue unchecked, however, a third method of defense is resorted to. This consists in a congestion, then an infiltration and finally an organization of a zone of tissue around the infected area which completely and more or less effectively prevents further spread of infection. In such event, even though a focus of infection has been formed, so long as the protecting wall which encloses it remains intact, the patient is reasonably free from the likelihood of secondary involvement, and hence may suffer from nothing more than the local lesion.

It is precisely because of the defense mechanism with which the tissues are endowed that escape from general infection ever takes place in any individual after once bacteria pass the surface epithelium.

Since the mechanism of bodily resistance inherent in the tissues varies greatly in different individuals, it follows that one who is normal in this respect may escape infection entirely, even during severe epidemics; another whose physical powers are re-

duced by disease or disturbed nutrition becomes locally infected, while a third, whose resistance is greatly lowered will, in the presence of virulent organisms in great numbers, almost certainly suffer from both focal and secondary general disease.

In a majority of all instances of focal infection in which the process is carried to the point of being well walled off, further spread of the disease never takes place, or at least in most cases not for a long time. Even after leakage of toxins from a focal site into the blood stream has occurred, the combative elements of the blood continue active in defense of the body and, no doubt, are often successful even then in crippling or destroying the bacteria, and thus modifying or preventing secondary general infection which otherwise would certainly follow. Granting this, it is still certain that such happy ending is not always possible for clinical experience teaches that the toxic contents of the primary focus may enter the system at any time even though the affected area seems well isolated by a protecting wall.

Spread of infection from a focal site to the system occurs either through the lymphatic system or blood stream, and often, no doubt, through both channels in the same individual. Of the two routes, dissemination through the blood occurs oftenest. It is believed by clinicians and pathologists that the more distant tissues and organs of the body receive infection almost wholly through hematogenous channels, whereas the lymph nodes and nearby situations more often are involved through lymphogenous sources. The lymph structures of the head, neck and mediastinum are always, no doubt, affected secondarily through the medium of the lymph stream. Infection, subsequent enlargement and even destruction of the nodes of the head and neck are invariably caused by direct transmission of infecting agents from the ear, mouth, tonsils, paranasal sinuses or other structures of the head. Foci of infection in any of these tissues is almost always followed by secondary involvement in some degree of the lymph nodes of the pharynx and neck. Crowe found in a critical examination of one thousand cases of chronic tonsillar infection that more than one-half the number had enlarged lymph nodes at the angle of the jaw. Crowe does not state the condition of the postpharyngeal glands in these cases, but it is known that these are often first affected.

Lymph nodes, like other body tissues, are well provided with defense organs against bacterial invasion. Through such de-

fense the infection is often overcome in the first nodes attacked, and the invasion comes to an end. In other cases many nodes are invaded before the defense is sufficiently powerful to stop the progress of the infection, and some of the nodes may be broken down and destroyed in the effort. In yet more violent cases of infection the toxins may pass through the whole chain of nodes and ultimately reach the blood stream, in which event systemic infection takes place exactly as if it occurred through hematogenous channels originally.

Infection reaching the heart, kidney and other vital organs is carried almost exclusively by the blood. The method by which the bacterial toxins and the bacteria themselves reach the blood or lymph streams is chiefly through a process of thrombosis of the capillaries which surround the affected area, and the subsequent discharge of emboli directly into the circulating fluids.

The behavior of the bacteria during their residence in a focus of infection is of interest. Rosenow and his co-workers have apparently proved by extensive animal experimentation that organisms, chiefly of the streptococcus-pneumococcus group may undergo a change or transmutation during this period, and that in assuming the new form they also acquire an elective affinity which enables them to choose, as it were, which tissue of the body they shall attack when once set free into the general circulation. It is because of this change in the nature of the organisms and their acquired affinity for a given organ of the body, it is claimed, that the heart is attacked in one instance, whereas some other tissue suffers in another. Billings, working with Rosenow, but from the clinical standpoint, has confirmed Rosenow's findings in a large number of cases.

Holman is not in complete accord with Rosenow's conclusions, but does agree that it is definitely demonstrated that stereptococci localize in the various organs of the body, and that such focalization produces serious disease. Kolmer thinks the question of the selectivity of bacteria in attack on bodily tissues has been overemphasized,—an opinion with which other bacteriologists seem sympathetic. It matters little in a practical way, however, whether one or the other view is correct, but the fact that all are agreed that secondary general infections originate chiefly in the head matters much, for it is evident that if the primary seat of a disease can be determined, treatment of the sec-

ondary affection at once becomes greatly simplified. The primary focus is therefore of paramount interest, and the determination of its presence a first requirement in the management of secondary general diseases.

Since the majority of all foci which lead to secondary infection and systemic disease are situated in the head, internists into whose care such patients usually fall very properly insist that the otolaryngologist determine the presence and extent of the primary seat of the lesion. It is beyond the scope of this paper to discuss either diagnosis or treatment, except in a general way, but it is undoubtedly proper to point out and emphasize the necessity for patience, thoroughness, and the use of every modern means in the effort to determine with precision all possible facts concerning the origin of the systemic ailment. It should also be pointed out and emphasized that failure to benefit or relieve the systemic disease may result from misinformation based upon a hurried, incomplete examination, which may be given the internists. Complete examination of the usual sites of foci of infection in the head should be made even though the internist has already located a focus, (as the appendix or gall bladder), in some other part of the body, for it is known that two primary infected areas may be present in the same person at the same time, and that foci outside the head are often secondary. In either event it is evident that treatment of one source, while overlooking the other, would end in failure to cure.

In a majority of instances in which primary infection in the head is followed by systemic disorders, it is not difficult to determine either the presence or site of the primary lesion. Abundant, or even moderate discharge of pus from the ear or paranasal sinuses will be recognized readily, and palpably diseased teeth or tonsils will easily be seen and their influence on the general disease properly assigned. Other cases are not so easy. In many instances in which secondary systemic disease has developed from a chronic focus, no focal area can be found in the head for the reason that healing has taken place after the secondary involvement occurred, and little or no evidence of the primary lesion may remain. In other cases the primary focus may be quiescent at the time of examination, and if so, symptoms both objective and subjective may be wholly in abeyance. Then there is another type in which there is no physical evidence of suppuration, or at most but a trivial amount

of visible pus, even though the focal disease is active. Cases such as these are common in relation to the teeth, tonsils, mastoid cells and middle ear, and the paranasal sinuses, and the observation has been repeatedly made by clinicians that it is in the type of unsuspected primary lesion that many of the worst secondary afflictions occur. This opinion is held especially concerning infections that take place at the roots of devitalized teeth, in which no apical abscess is present, but from a zone of infected tissue surrounding which pathogenic bacteria of a low degree of virulence may almost always be isolated. While the teeth and tonsils have been held responsible for systemic disease in many cases where no palpable abscess can be determined, it is equally certain, and has been proved clinically, that other foci of the head equally difficult of demonstration, are as frequently culpable.

In all instances such as the foregoing, the difficulties attendant upon definite diagnosis are obvious, but bearing in mind the presence of a systemic disease in any case, it is equally obvious that efforts to determine the exact origin should be thorough and inclusive. A safe rule is to make more than one examination in all doubtful cases; to secure the co-operation and judgment of internist, dentist, roentgenologist, and sometimes a laboratory expert, before a final decision is announced. In all chronic systemic affections a complete history of the case is of great importance. Such a history will often give information relating to ear or nasal diseases, or affections of the teeth or tonsils, that have long been forgotten by the patient, but are of great aid in diagnosis.

It is found in many instances that secondary general infection, if chronic, often has become a disease entity itself. The primary focus has done its mischief and has wholly subsided, but the secondary disease continues on indefinitely. New foci in distant organs—the heart, kidneys, lymph nodes and other structures—are set up, and these secondary areas of infection feed their toxins into the system as certainly and as harmfully as did the original primary focus in the head, even during its period of greatest activity.

It is undeniable that recognition of the true relationship between primary focal infection in the head and subsequent secondary infection of the body has not always been adequately

recognized, and that this view is especially true in cases of foci that are not active, are quiescent, or have completely healed.

Before any diagnosis can be considered definite, it must be based upon searching inspection of all regions in which foci of infection commonly occur. Should an evident focus be found during the search, it would be manifestly unwise to conclude that the sole pathway of bacteria into the system had been found, and terminate further search, for on complete investigation it is often proved that more than one active focus is present in the same person.

Numerous estimates have been made concerning the number of general affections that have definitely been traced to a focal source. As many as seventy have been given. The number is undoubtedly large. Evans believes that the following diseases have such origin:

Acute endocarditis	Acute bronchopneumonia
Acute pericarditis	Acute cholecystitis
Acute pleuritis	Acute appendicitis
Acute peritonitis	Acute enteritis
Acute arthritis	Acute colitis
Acute tenonitis	Acute gastic and duodenal ulcer
Acute bursitis	Acute conjunctivitis
Erythema nodosum	Acute keratitis
Purpura hemorrhagica	Acute iritis
Acute iridocyclitis	Acute pancreasitis
Acute uveitis	Acute nephritis
Acute episcleritis	Acute pyelitis
Acute choroiditis	Acute neuritis
Acute retinitis	Acute myelitis
Acute optic neuritis	Acute polyomyelitis
Acute thyroiditis	Acute meningitis

Herpes zoster
Acute chorea
Acute myositis
Acute myocarditis
Acute osteomyelitis

And to these should be added lymphadenitis, which is of frequent occurrence as a result of the same cause. Many of the above affections become chronic and persist through life.

There is little question concerning the necessity of including treatment of the primary focus as an essential part of treatment of the secondary general disease, for it is evident that so long as leakage from the focus continues to pollute the body fluids, little can be done to arrest the secondary affliction. The most effective methods of dealing with the primary focus is to completely eradicate it. Methods of eradication differ. Good drainage may prove sufficient in some cases, while surgical eradication is essential in others. Thoroughness in the sense that complete removal of the infected area by surgical or other means is often impossible because of the wide extent or dangerous location of the primary lesion.

The best method of treatment, should opportunity be given, is prevention. Prevention of the initial infection in the head is best assured by securing adequate drainage of all cavities abnormally predisposed to stagnation of secretion and favorable to lodgment and growth of bacteria, and the removal of diseased tissues like the tonsils that contain crypts which also favor the lodgment and propagation of disease germs. Measures such as these are, of course, carried out at a time previous to the establishment of either the primary foci or the secondary general complication.

While the relation of focal infection to general disease was recognized long ago, it is only during the past three decades, as previously stated, that the question attracted the keen interest it deserved. During this period a great number of operations have been performed with a view of eradicating the primary seat, and of curing the general disease, should one be present. Sufficient time has now elapsed to justify the collection of statistics relating to the effect these surgical procedures have had upon the amelioration or cure of secondary diseases. Many statistics covering a large number of cases and collected from various sources do not show satisfactory results from operation, or at least not insofar as good effect on the general disease is concerned.

Dick, in a careful resume of the subject, states that the statistics of operations are not universally good. Hunt says that tonsillectomy has little effect on the course or relief of acute rheumatic fever. Many other observers are equally pessimistic concerning the good effect of operative measures. Kaiser, for instance, reports a study of 48,000 school children in an attempt

to arrive at some definite conclusion. Twenty thousand of these had their tonsils removed about five years previously, whereas 28,000 had not been operated. Comparing the incidence of subsequent general disease in each group, he found that eight per cent of those who had been operated continued to have joint pains, growing pains or rheumatic fever, whereas those not operated had the same symptoms of general infection in only ten per cent,—a difference too small for encouragement. He found also that in chorea four-tenths per cent of those operated continued to suffer from the affection, while of those not operated only five-tenths per cent continued to have the disease, showing again but slight difference. Of the operated cases 2.2 per cent were affected by rheumatic carditis, while only 2.9 per cent suffered from the same disease among the unoperated,—certainly not a startling contrast. Fein, a German writer on the subject, goes so far as to consider recovery from general infection more certain without *than with* operation on the primary focus. Judging by the above figures, it would appear that those who are operated for the removal of foci of infection were but little better able to resist secondary infection than those not operated.

On the other hand, those who have reported the results of their own operations, or have estimated the findings in cases of which they have personal knowledge rather than promiscuous, statistical information, speak more encouragingly. Thus, Faught thinks operative measures directed toward the removal of infected foci are almost universally good. Holman states that the effect of tonsillectomy on acute rheumatic fever should be considered satisfactory. Lambert asserts that it has been his experience that rheumatic fever was benefitted by tonsillar and dental operations, and Mackie, who studied the course of rheumatic fever in 393 cases and estimated the results of operation on the primary foci, believes that the tonsils are a frequent cause of the affection and that their removal is beneficial. Mackie, therefore, advocates tonsillectomy in all cases where it is definitely shown that the tonsils are affected. Robey and Friedman hold a similar belief and advocate complete eradication of tonsillar infection as the best mode of treating acute rheumatic fever and the secondary inflammatory affections of the heart which often accompany it. Daland is positive that eradication of the primary focus is essential and often curative.

Marriott and Jeans have found that infants often suffer from unsuspected infection of the mastoid or nasal sinuses and that these foci give rise to serious nutritional disturbances and intestinal inflammation. In such instances Marriott states that removing the foci relieves the secondary complications. Dean, Alden, Proetz and others have confirmed Marriott's statements clinically on many operated cases.

Crowe has reported the effect on general disease of the removal of infected tonsils in one thousand cases. Many of these were re-examined in from one to three years after operation and the condition of the patient then was carefully compared with that at time of operation. The conclusions gained from a study of his statistical tables indicates that unquestioned benefit and frequent cure of the systemic disease followed eradication of the foci in those who suffered from enlarged cervical glands, infectious arthritis, rheumatic fever and nephritis. Many of the reported cases having acute rheumatic fever also had at the same time some type of inflammatory affection of the heart, and, while the heart disease continued after operation, it may be inferred from the report that there was likewise much improvement of the cardiac disease after the tonsillectomy.

The result of Crowe's operations for the relief of chorea was disappointing and in this respect confirms the statistics on that subject already quoted from Kaiser. Crowe also reports unsatisfactory results in cases of operation where rheumatoid arthritis is present. Benefit was often noted after removal of the focus but the arthritic recurrences were frequent and sometimes the ailment was worse after than before operation. It should be noted that while the title of Crowe's reports suggest that operation in the one thousand cases was confined to the tonsils, the tables show that in many cases nasal, nasopharyngeal and sinus operations were also done at the same or some subsequent time.

The diversity of opinion given above would be confusing and would lead to uncertainty as to the best methods of treatment unless carefully analyzed. Mass statistics compiled from examination of school children and from sources representing promiscuous surgery performed everywhere, by every type operator and under all circumstances as to environment and after care, cannot be reliable and should not be accepted as a basis on which to judge the results of surgical removal of primary foci of

infection on secondary general diseases. Statistics of this kind mean nothing more than that a certain number of individuals were operated in some unknown way and that certain effects were noted. Oftentimes the objects of these statistics were not re-examined by competent observers. Often the patient's word is accepted that he was completely operated but not cured. Little information is available in such statistics to indicate whether or not the operation was thorough and included all the primary foci. Little is stated to indicate whether or not the patient operated was examined for foci other than the one on which operation was performed. The inference given out by such tables is, of course, that all cases were thoroughly operated; that failure ensued, and that therefore operative measures are useless. Such is evidently not the case, for re-examination by physicians who have been painstaking in the use of all modern methods has shown that many who have been reported as having undergone operation were in fact but inefficiently operated. Tonsil stumps were left, infected teeth remained untreated, and other foci had been entirely overlooked.

Of interest in this connection, Brock has stated that of eighty cases of tonsillectomy performed for the relief of arthritis, forty-two still had infected stumps remaining. And Dick says that whereas a study of the statistics of results from operative procedures intended to eradicate primary foci and cure general disease are not good, he cites as a reason the fact that tonsil stumps often remain, tooth roots have been overlooked, and that infected sinuses or other foci have been but partially cleared of infection. Dick actually found in a series of clinical and laboratory investigations that infected tonsil stumps were still present in 290 of 403 operated cases. He also makes the observation, which has been confirmed by many others, that tonsil stumps which remain after operation contain more infectious material and are therefore more subject to inflammation than they were previous to the operation. Payson Clark makes a similar statement and reports that of 143 cases which he examined because of subsequent complaint after tonsillectomy, badly infected stumps still remained in fifty-nine. Crowe's statistics on the question are interesting in this connection. His tables showing results in one thousand cases indicate in many instances that failure to relieve or cure the secondary general infection was due to the fact that the patient carried other foci

than the one on which operation had been done, but would permit no further surgery.

Doubtless most otolaryngologists have reached similar conclusions to those here quoted, as to the reasons for operative failure. Certainly my own experience bears out the belief that benefit to the general ailment follows the removal of an active primary focus, and that failure is often, or usually due to failure on the part of the operator to include removal of all primary foci, or to the fact that the secondary disease was already well established.

Of course, no one will deny that failure to relieve or cure systemic disease of focal origin sometimes occurs after complete removal of all focal causes in certain types of cases, or that such patients occasionally are made worse, at least temporarily by surgical interference. But it should be said in fairness that an examination into the pathology present in such persons often furnishes an entirely rational explanation for the failure. Take for example a case of cardiac complication in which the primary focus of infection situated in the head is still more or less active. Assume that the heart is already seriously crippled, as it often is, and that secondary foci of infection are already established in the endocardium. In every such instance the secondary disease is already an independent disease and will continue on, fed as it were by toxins from its own well established secondary focus of infection. Removal of the primary source in the head at this late period may be of benefit only in exact proportion to the amount of leakage into the system which still goes on from that source. In other instances, it is impossible to remove by surgical or other means, all the primary areas in the head for the reason that to do so would lead to dangers wholly unjustified. This is especially true of infections of the ethmoid cells and sphenoid sinuses, and in pansinusitis where operations even approaching the desirable degree of thoroughness might invite intracranial complication of a serious type.

It must be evident therefore that after secondary lesions are well established and pathologic changes of a permanent nature have taken place in distant organs, or when circumstances indicate that it is unwise or impossible to remove the primary foci completely, it is unreasonable to expect relief or cure from surgical procedures, and that other means of treatment must be relied upon. Treatment of the patient himself is often valuable

in such instances. Indeed, many circumstances in the lives of those suffering from the secondary effects of focal infection may be such that failure to cure or relieve is inevitable, even though all sources of the infection have been completely removed in favorable cases, unless the patient himself is taken into account. Because of the infection all such individuals suffer from lowered resistance; therefore their physical condition is easily and frequently disturbed. Their environment and hygienic status may be bad; economic necessity may drive them to tasks too severe; questions of proper food, air and sunshine may need direction; and, above all, especially in cardiac complications the question of rest in bed is paramount. All these items essential to physical betterment are problems for the internist, whose co-operation in all cases of general, secondary infection is wholly essential. Working together patiently and in harmony, the combined labors of otolaryngologist and internist are often rewarded by commendable, if not brilliant results.

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DISCUSSION.

DR. JUDSON DALAND, Philadelphia: It was indeed a great pleasure to accept the invitation to meet with you today and discuss Dr. Barnhill's excellent paper, so full of valuable suggestions.

Focal infection is as important to the physician as asepsis is to the surgeon. Too much emphasis cannot be placed upon focal infection as a cause of systemic disease, and a list of these diseases would resemble the table of contents of a textbook on the practice of medicine. Usually a focus of infection is composed of a mass of bacteria and toxins, and an amount no larger than a split pea may cause any one of the diseases secondary to focal infection. The diagnosis of a latent focus of infection is always difficult and occasionally impossible. Multiple primary foci of infection are common.

A closed focus of infection is one shut off from air, and the best examples are: periapical infection, an infected tonsil or remnant with crypts closed by adhesive inflammation; or a closed cell in parasinusitis sinusitis. When a closed infection occurs it is dangerous, as an avirulent streptococcus becomes virulent, pathogenic and selective, and if *drained* and *aerated*, may again become avirulent, due to variation in oxygen tension. As a rule, no pus is present.

The daily absorption of small quantities of toxins may cause immunity, thereby suppressing symptoms. This immunity may be lost by many causes such as intercurrent disease, accidents, insufficient food or sleep, excessive work, exposure, or old age. Occasionally decreasing immunity may be detected, in the absence of symptoms, by the presence of lymphocytosis, with a corresponding decrease in the polymorphonuclear cells, and leukopenia.

Periapical infection may cause chronic tonsillitis by the way of the blood stream, and pyorrhoea, especially around the last molars, may cause tonsillar infection by continguity. An infected tonsil may cause periapical infection. As a rule, chronic or latent tonsillitis causes toxemia, and later bacteria, in numbers or as an embolism, may enter the circulation. Frequently, toxins and bacteria enter the blood intermittently, alternately or simultaneously. Toxemia is probably more frequent and continuous than bacteria. Toxins possess varying degrees of virulency, pathogenicity and specificity.

In the diagnosis of latent tonsillitis, importance is to be attributed to a history of diphtheria, scarlet fever, streptococcic sore throat, or one or more attacks of tonsillitis in childhood. In certain cases the original infection of the tonsils may have occurred from one to twenty-five years previously. I incline to the opinion that most tonsils when once infected remain infected.

A study of 672 cases of focal infection, chiefly observed in consulting practice, showed that nearly all of the primary foci of infection are in the head cavities—25 per cent involved the tonsils alone; 23 per cent involved the tonsils and teeth; 6 per cent involved the tonsils and two other foci, and 8 per cent involved the tonsils and three other foci of infection. The percentage of tonsillar infections associated with other foci was the same as dental infection with other foci, i. e., 61 per cent.

Difficulties of diagnosis are best illustrated by the following brief abstracts of two cases:

Male, aged 22; tonsils and adenoids removed at seven; septic fever at ten; broncho-pneumonia and appendectomy at twelve; recurring attacks of septic fever at the ages of thirteen, fourteen and seventeen, erroneously diagnosed influenza; submucous resection of the septum and catarrhal jaundice at twenty-one. These diseases and operations were due to an undiagnosed focus of infection.

Examination revealed myocarditis, emaciation, weakness of the nervous and muscular systems; intestinal toxemia; chloroanemia; extreme lymphocytosis (39.5 per cent.) The tonsils had been completely removed and there was no evidence of infection of the teeth, gall bladder, prostate or elsewhere. The diagnosis of sinusitis was made by exclusion. Dr. Ross H. Skillern reported that he found no evidence of sinusitis and was advised to explore. He removed the softened and diseased walls of the ethmoid, evacuated a pocket of pus near the cribriform plate, which was soft and diseased and when removed exposed a normal dura matter. The patient made a good recovery.

Spinster, aged 69; emaciated, adynamic, neurasthenic, apprehensive of death, tachycardia, subconjunctival hemorrhages, chloroanemia, extreme lymphocytosis, and leukopenia. Several infected teeth were extracted, followed by severe hemorrhage. Improvement, aided by an autogenous vaccine, continued progressive for seven months, and was then followed by a relapse. Reexamination revealed no focus of infection. The tonsils were small, buried and invisible, and although there was no history of previous disease they were diagnosed, by exclusion, as being infected. This patient was an extremely hazardous surgical risk. Nevertheless, Dr. Ross H. Skillern courageously and skilfully removed the tonsils, which were diseased, the infecting organism being the streptococcus hemolyticus. Subsequently she steadily improved, regained a fair degree of health, and the chloroanemia, lymphocytosis and leukopenia disappeared.

The treatment of chronic tonsillitis or sinusitis is surgical. Occasionally the results of complete removal of infected tonsils are miraculous, as for example: the disappearance, over night, of cardiac arrhythmia of several months' duration; of long continued pain in peripheral nerves; or of pain and dysfunction of the left hip joint lasting a year. It is evident that these cases are chiefly toxic. Improvement usually is steadily progressive, and may be accelerated by an autogenous vaccine.

Occasionally secondary bacterial infection occurs in the endocardium, gallbladder, joints, or elsewhere, and continues as an independent focus of infection after the complete removal of the primary focus.

Failure to secure good results is usually due to: removing one focus and allowing one or more to remain undiagnosed; incomplete operation upon the sinus; allowing a remnant of infected tonsil to remain after a tonsillectomy; a secondary bacterial focus behaving as did the primary. The importance of drainage in sinusitis is recognized, but the great value of aeration is occasionally forgotten.

The profession is in great need of additional diagnostic signs of chronic ethmoiditis and sphenoiditis.

DR. T. E. CARMODY, Denver, Colorado: It is very hard to discuss a paper of this kind, especially since it has been covered so completely by Dr. Barnhill and Dr. Daland. We all know the work of these men.

One of the important things that we must consider is the point of incipiency of the disease—where does it originate? I believe that most of

the infections and infectious diseases with which we have to contend originate in the mouth, nose and throat, and we have the authority of Dr. Charles Mayo that 85 per cent of all disease originate above the collar bone. Whether that is true or not, we like to believe it, and I think it is largely true.

How are we going to tell when we have incipient disease? The trouble is, from the standpoint of the laryngologist and also of the internist, that we get the patient after the infection has been well engrafted into the body. We see the sinus infection after it has been going on for months, and often for years. We must begin with the infant at time of birth and see if we cannot keep it from getting the infection. Whether we can or not remains to be seen.

The number of heart affections that we have is indicative of the number of focal infections we have that become secondary. The primary focal infection of course is the one we should prevent. If we have a primary infection and then get a secondary infection, then our eradication of the primary focus as Dr. Daland has just said, must help the secondary condition, only by preventing further dissemination.

Many of our operations, as Dr. Daland has aptly said, are late. Dr. Barnhill also calls attention to that. And many are incomplete. I do not think there is any question about that. If we can only complete our operations we would probably prevent a great deal of secondary infections.

Another point that I belive is in Dr. Barnhill's original paper, is that we cannot eradicate all of our primary infection because there is a possibility that we have two or more primary foci. Whether the primary focus occurs in the tonsils, in the teeth, in the sinuses, or elsewhere, makes very little difference. We may have it in all three, or even more. Dr. Beck some years ago called attention to the fact that we may have many primary foci of infection, and on trying to get the percentages, as Dr. Daland has given us, we find in some cases only one, and in some others two or more. We cannot always eradicate all of these, generally for the reason that the patients are poor surgical risks, some of them so poor that we are unable to eradicate even one primary focus.

I was glad that Dr. Daland called our attention to the point that focal infection, from the standpoint of the internist, stands in the same relation as asepsis from the standpoint of the surgeon. We all agree with him on that.

The infection of the teeth—shall we consider it primary to the infection of the tonsils or sinuses? That is a point that has been of great interest to me. I believe infection of the sinuses is primary in most cases, and the drainage is downward. Many of us were taught in years gone by in regard to infection of the teeth, that we must take into consideration that the teeth are not decayed and we do not have pyorrhoea under twelve years of age, and there is not much chance of infection of sinuses from the teeth under that age. There may be, as Dr. Berry has brought out, a great deal of infection later in life, and of that we have no doubt. But the infection primarily is in the sinuses and we have seen some sinuses infected under one year of age, and we have one case at eight days of age. If we have infection that early we will have secondary

changes so early in life that there is no question but what our heart infections are secondary to infections of the sinuses or tonsils—it makes very little difference what the cause, we get them too early.

Another thing we must consider is the possibility of infection both from the blood stream and through the alimentary tract. We may have infections passing through the stomach, gallbladder and appendix, both from the bloodstream and from the alimentary tract. We have the same thing in pulmonary infections. They may be both aspirated, and come from the bloodstream.

There is one more point, and that is the vaccines to which Dr. Daland has called our attention—the use of autogenous vaccines. We all use autogenous vaccines, and we may get some results, but I do not believe the ordinary stock vaccines are worth anything, except as foreign protein.

Dr. Eugene R. Lewis, Los Angeles, California: This is an unusually valuable paper. It requires re-reading and slow digesting. In general I am in accord with Dr. Barnhill's statements, and even more so with his attitude. When he says: "Primary focal infection is responsible for all secondary diseases of an infectious nature," I feel that I would put it somewhat differently, although it may be that even then Dr. Barnhill and I are in accord. I still adhere to the belief that much of the effort expended in identifying and killing bacteria encountered might be more profitably directed to studying and attempting to rectify the biochemic and biophysic alterations in the host. However, I am not here to discuss that further.

Much of the responsibility for infectious disease lies in the prime or determining condition or combination of conditions which made possible the onset of the infection. Dr. Barnhill observes, quite properly, that bacteria of different varieties and degrees of pathogenicity inhabit body-surfaces for long periods without the incidence of infection. Some determining cause—trauma, exposure, fatigue, emotion, or mechanical or physical disturbance, affords opportunity for bacterial infestation to be converted into bacterial infection. Until the tissue conditions underlying that conversion from infestation to infection have been reversed, at least one of the prime causes continues to operate. This then, constitutes a pregnant suggestion as to one of the first and most important attempts of treatment, namely, to reverse, or attempt to reverse, those tissue conditions which determine the onset of infection. Success in reversing these conditions may still leave here and there foci more or less completely surrounded by organized exudate. These foci remain a problem after the main infection has disappeared. The obvious optimum therapeutic effect in dealing with foci must follow stripping them of their protective capsule, permitting continuous access of natural anti-infectious agencies to these foci. Complete recovery in this manner offers maximum conservation of essential tissues.

Dr. Lyman Richards, Boston, Massachusetts: In connection with the subject of focal infection I should like to mention a question of vital interest to those of us connected with institutions which treat children. A never-ending source of difficulty is the so-called tonsil clinic to which are

referred large numbers of children for a decision as to the advisability of the operation of tonsillectomy. Coming to such clinics are always a certain number in whom one look into the mouth is sufficient to decide the question. They do not all, however, fall in this class. Many of them are referred either by pediatricians, school doctors, or health nurses for a wide variety of ailments ranging from anorexia, malnutrition and weakness to growing pains and enuresis.

Shall these children be subjected to tonsillectomy for such indefinite complaints, and how are we to decide this question by nothing more than the usual laryngological examination? By what standards, on the other hand, shall these children be excluded from operation, regardless of the wide range of their symptoms? Who can say for a certainty by any sort of throat examination that they do or do not require tonsillectomy?

This situation has so distressed us at the Children's Hospital in Boston that we have been forced to evolve some more satisfactory method for handling it. Heretofore the vast majority of these so-called medical complaints were hopefully subjected to tonsillectomy, unless there was some obvious and clear-cut contraindication to operation, discovered only by the preoperative physical examination made by the surgical interne. Of late we have added to our laryngological staff a medical interne trained primarily in pediatrics, one of whose duties it is carefully to examine each child accepted for tonsillectomy. This examination is no hasty routine passing of the heart and lungs, but is an honest attempt to discover if possible what other ailments besides the suspected tonsils may be at the bottom of these vague symptoms. Granted that tonsillectomy is advisable, granted that it will not do any harm at the worst, what else may the child have which, if unrecognized, might lead to the persistence of these same symptoms and hence to an operative failure? This is an important question to answer.

Where such a real physical examination discloses conditions which should require attention, the operations may well be carried out, but the child is marked as one needing further observation, perhaps even admitting to the ward for further study, treatment, more thorough blood studies and urinalysis. In short, any one of the group of medical procedures which may insure ultimate recovery without which a simple tonsillectomy, advisable in itself but not a panacea for all ills, would be a complete failure.

In this way we know far more about the patient on whom we are operating and our chances of curing symptoms of more obscure nature than the classical indications for tonsillectomy are far better.

DR. R. H. T. MANN, Texarkana, Texas: I have very much enjoyed Dr. Barnhill's paper. He has brought to us a very interesting subject, one about which there is so much yet to be learned. There is one phase of this subject, however, that he did not mention, and with which I have now had sufficient clinical experience to recommend its trial, at least in a certain number of well selected cases. That is the removal of the focus of infection during the acute febrile attack of the disease, but after the acute inflammatory process in the original focus has subsided. Let me illustrate what I am trying to say. A patient has an attack of tonsillitis;

in a week or ten days he develops an acute arthritis with all the febrile symptoms accompanying this disease. Now if a tonsillectomy is performed on this patient as soon as the tonsillitis has subsided, but while the acute arthritis still persists, it is remarkable to note in many such cases the very rapid recovery which follows. It seems, therefore, to be clinically true, at least in many of these cases, that the original focus, although the inflammatory process has subsided, is still producing all the symptoms in remote parts of the body and not the development of new foci in these parts, because as stated, when the original focus has been removed a rapid recovery ensues, which was not the case until this was done.

DR. JOHN F. BARNHILL, closing: I wish to thank the gentlemen who have spoken for emphasizing so many essential points in my paper. I also wish to emphasize a few.

We are hearing much from certain internists and statisticians to the effect that operative measures directed to the eradication of primary foci in the head too often have little or no effect on the relief or cure of secondary general affections having their origin in that region. I pointed out in my paper, and wish here to repeat, that most available statistics on the subject are unreliable. It must be admitted, of course, that failure to relieve general affections by operative measures on the primary focus is sufficiently common. However, investigation usually discloses reasons for failure that could not have been avoided. It is wholly unreasonable to expect cure or even great relief from surgical methods of treatment: first, after hasty and therefore unreliable diagnosis; second, after incomplete eradication of the primary focus; third, after a secondary focus has been well established in some distant organ, as in the heart, kidney or joints.

A study of recent published statistics, even unfavorable as they seem, have not shaken my faith in the efficacy of surgical methods, especially when employed early and based on painstaking, accurate diagnosis.

LABORATORY AIDS IN EAR, NOSE AND THROAT CONDITIONS

CHARLES E. CONNOR, M. D.,

MILLER CLINIC,

SAINT PAUL, MINNESOTA

Blood studies are capable of yielding a wealth of information in diseases of the ear, nose and throat. The diminution in red cells and hemoglobin in sepsis, especially of hemolytic streptococcus origin, has long been known and can be made use of in gauging the status and progress of severe septic aural processes; the loss of red cells may vary from 200,000 to 1,000,000 per week and they may drop to 1,000,000 in a few days.[1] The loss of hemoglobin may parallel or outstrip that of red cells. Kopetzky[2] finds such studies of great value, especially in cases of hemorrhagic mastoiditis, due to the streptococcus hemolyticus. The progressive loss of red cells and diminution of hemoglobin in a case of otitis media with septic temperature suggests such a lesion and may offer early operative indications. A favorable postoperative course is attended by gradual increase in red cells and hemoglobin; should progressive loss continue extension into the lateral sinus is to be suspected and a transfusion of whole blood given. If the reduction still continues the aural infection should be eliminated as far as possible from the general blood stream by suitable surgical measures.

Infection changes the white cell picture markedly. There is usually an absolute and often relative increase in the neutrophils; eosinophils and basophils diminish or disappear and lymphocytes are much diminished. During convalescence the neutrophils gradually return to normal, the eosinophils reappear and the lymphocytes increase until they may even exceed the normal number, the post-infectious lymphocytosis. The monocytes also show an increase during convalescence.

The total white count and percentage of neutrophils has long been accepted as a guide to the reaction of the patient and the

severity of the infection. This is especially true in acute sepsis, in which the leukocytosis may vary from a normal of 8,000 to 45,000 or 50,000 or more. Wicart[3] found the following values in aural sepsis:

	White Cells	% of Neutrophils
Acute suppurative otitis media	6,000 to 10,000	70 per cent
Acute suppurative mastoiditis	10,000 to 12,000	72 per cent
Thrombophlebitis	20,000 to 25,000	92–95 per cent
Circumscribed meningitis	20,000 to 25,000	94 per cent
Diffuse meningitis	35,000 to 60,000	92–95 per cent
Cerebral abscess with encapsulation	20,000	88–95 per cent

Relatively few acute infections, such as malaria, typhoid, influenza, measles, and mumps, are attended by leukopenia. Leukocytosis depends on the resistance of the patient and the severity of the infection. When resistance is good and infection severe leukocytosis may be early and pronounced; if these two factors are less marked but proportionate the reaction is less sharp, comes later, and disappears sooner. If resistance is poor or infection overwhelming an actual leukopenia may develop, well illustrated by agranulocytic angina. Successful surgical intervention in sepsis should be attended by a decrease in the total white count and percentage of neutrophils; thus Schmidt[4] found a marked diminution in the leukocytosis of peritonsillar abscess, which often ranges from 15,000 to 23,000, after incision, while interference in pharyngeal phlegmon caused no change. He considers the white count of more diagnostic significance in such cases than the temperature curve. Should leukocytosis and neutrophilia persist after surgical interference continued sepsis should be suspected; should these factors increase after having dropped, the onset of a complication should be thought of.

Study of the neutrophil nucleus as a guide to the progress of infection was proposed by Arneth[5] in 1904, when he showed that during infection cells with immature nuclei appear in the blood and disappear with convalescence. He at first divided the nuclear forms into five classes as follows:

1. Vesicular, round or slightly indented, or simple but deeply indented nuclei, 5 per cent.
2. Nuclei in two segments, 35 per cent.
3. Nuclei in three segments, 41 per cent.
4. Nuclei in four segments, 17 per cent.
5. Nuclei in five or more segments, 2 per cent.

A further subdivision of the neutrophil into 20 groups was made and a classification of lymphocytes and monocytes so extensive that the total number of divisions was eighty-one. There has not been wide agreement with Arneth's theory that the age of the neutrophil is exactly indicated by the complexity of the nucleus. The greatest difficulty with his classification was the practical one of recognizing minor variations in nuclear form when obscured by pyknosis or degeneration. The method was cumbersome and was not used extensively until Schilling[6] simplified it by using only four divisions, the myelocyte, normally seen only in bone marrow, the young metamyelocyte (W cell, jugendliche), rarely seen in the peripheral blood, the old metamyelocyte (T cell, staff cell, band cell or stabkernige), and the adult segmented neutrophil, which comprises about sixty-three per cent of the total count. This classification has made the procedure much simpler so that it may be used with but little loss of time.

Nuclear metamorphosis is indicative of the changes going on in the bone marrow. White cells come from three sources. Granulocytes (neutrophils, eosinophils and basophils) come from the bone marrow, lymphocytes from lymph glands and lymph marrow, and monocytes (large mononuclears and transitionals) from the reticulo-endothelial system. Infection or toxemia may influence the bone marrow, the effect ranging from slight or strong stimulation to injury or destruction. These changes are reflected in the type of neutrophil. Slight irritation produces an increased output of normal neutrophils; with stronger stimulation immature forms make their appearance until in extreme cases the mother cell, the neutrophilic myelocyte, appears.

In making the count the young forms are put on the left side of the page and the mature forms on the right. In infection the percentage of young cells increases, hence the "shift to the left" or "linksverschiebung" and with convalescence the shift to the right or "rechtsverschiebung." Arneth counted 100 neutrophils, his figures being therefore, percentages of the neutrophils, while Schilling counted each of his four classes while doing an ordinary differential, his results giving him percentages of the total leukocyte count. The result is known as the Arneth or Schilling Hemogram.

White cells play different roles in inflammation. The neutrophil is a very sensitive indicator of the presence and severity of infection and degree of resistance and has long been made

use of in the estimation of leukocytosis and neutrophilia. Return to normal indicates subsidence of infection but usually takes place later than the corresponding change in eosinophils, lymphocytes, and monocytes. A drop in the absolute number of leukocytes with persistence of neutrophilia and shifting to the left is a bad sign inasmuch as the decreased number of cells indicates lessened bone marrow reaction while the neutrophilia and shifting to the left show that the irritative lesion is still present.

Eosinophils respond to infection more exactly and quickly than do neutrophils; with its onset they diminish or disappear, and with its subsidence they reappear, and may be present in unusually large numbers, constituting the post-infectious eosinophilia. One must, of course, rule out helminthiasis, vagotonia, and eczematous conditions which of themselves produce eosinophilia.

The role of the basophil is not well understood.

Lymphocytes react quite differently from neutrophils. In the presence of infection the total number may vary from ten to eighteen per cent instead of the normal twenty-three to twenty-five. A lymphopenia, therefore, usually accompanies a neutrophilia and if it becomes marked and persists in the presence of acute infection, constitutes a bad prognostic sign. With the subsidence of infection, the percentage of lymphocytes increases and in cases in which the disease is being held in check, convalescence or chronicity, increases to normal or above normal and constitutes the post-infectious lymphocytosis, a condition which is especially noted in children.

The monocyte is usually increased at the time of crisis, dropping slowly during convalescence.

Schilling has divided the leukocytic response to infection into three phases. The first he calls the neutrophilic phase of resistance; it is characterized by neutrophilia, shifting to the left, reduction in the number of lymphocytes and monocytes, and disappearance of the eosinophils. The second stage is the monocyte defense reaction, characterized by increase of monocytes, return of eosinophils, reduction of neutrophils and persisting shift to the left. The third stage is the lymphocyte phase of recovery; it is characterized by shifting back to normal, by increase in the number of lymphocytes and eosinophils and by diminution in the number of neutrophils and monocytes.

The hemogram is used to determine the severity and progress of infection. Some writers find the so-called staff count (referring to the immature or unsegmented "staff cell," "stabkernige" or "band form") of more value than the ordinary white count and differential, and frequently follow the case by repeated differentials with only an occasional leukocyte count. Kopetzky[2] finds it of inestimable value, especially in border line cases, and recommends its more general use. He feels that fifteen per cent of staff cells means continued infection, that twelve per cent means operation, and that any figure below twelve per cent permits further observation. Gale[7] reports the result of 250 counts made in suppurative ear lesions and does not find the hemogram a reliable guide to operative interference.

Hesse[8] found the total white count markedly increased in acute suppurative otitis media and mastoiditis, the higher counts meaning probable bone involvement; shifting to the left may or may not occur in acute, uncomplicated mastoiditis. He was unable by this means to separate cases of acute, suppurative otitis media with mastoiditis from those without mastoiditis. The most valuable aid of the Schilling Hemogram is in the recognition of major complications, but a differentiation of complications is not possible.

Heidemann[9] feels that one must know quite definitely the limits of value of the blood picture in otogenous disease. One can not wholly differentiate a simple otitis media from mastoiditis, even though something may be learned of the severity of the infection; occasionally operative indication is given. The fact that the blood picture may be normal in severe mastoiditis must be borne in mind. Chronic suppurative otitis media makes no change in the blood although acute exacerbations show much the same picture as an ordinary acute infection. There are no changes in otosclerosis.

Heidemann draws the following conclusions: occasionally the blood picture may offer operative indications in borderline cases but it is not of great importance in the average case of mastoiditis. It is more important in detecting the presence of intra-cranial complications. A normal blood picture in such a case means nothing but a pathological picture is an infallible sign of complication although differentiation is not possible.

Balden[10] is in essential agreement with Heidemann. She states that infections of the ear have no specific blood picture

and that differential diagnosis is possible only on the basis of marked changes in but few cases. Intercurrent infections, particularly grippe, obscure the findings; one must always take into account the age of the process, and the age and constitutional diathesis of the patient. Old lesions show a lymphocytosis, children have the same tendency, and certain patients, notably those with the so-called lymphatic diathesis, show the same thing. Gross disturbances usually register in the blood picture before clinical signs but in very acute severe cases signs and symptoms occur at once. Therefore, the blood picture is apt to be of more value in the border line case where slight deviations sometimes offer operative indications. Temperature, blood picture and clinical course correspond well. One can differentiate between otogenous infections in degree only and not with any marked accuracy. The presence of a complication may be detected but not its type. The blood picture is corroborative but not a determining factor.

Glasscheib[11] is one of the few writers who feels that he can draw very definite conclusions as to the exact state of the pathological process. In a study of 72 cases of suppurative mastoiditis he differentiated three groups:

1. Cases which had normal eosinophil percentage and a deviation index of 1-10 to 1-8 showed in the mastoid cells granulations but no free pus. (Deviation index is relation of immature cells to total neutrophils).

2. Cases without eosinophils and with a deviation index as high as 1-3 showed in the cells free pus and granulations.

3. Cases with eosinophils increased from 3-6 per cent with a deviation index of 1-3 or 1-2 showed pus-containing cells in contact with the sinus. A thrombosis is very apt to show a moderate grade of eosinophilia but one must rule out all other conditions which may cause eosinophilia.

Perisinus abscess shows a neutrophilia of from 85-90 per cent with a deviation index as high as 1-3. Sinus thrombosis shows an eosinophilia with a rise of the index to 1-2 or 1-1, and may be excluded when these two characteristics are absent; when it makes its appearance there is a specific decrease of neutrophils and leukocytes; therefore, the total leukocyte count is of no importance. Complications in the healing of the wound, such as phlegmon, erysipelas, and abscess do not show an index as high as 1-2. If the number of neutrophils rises to 85 or 90 per cent

or if the index becomes reciprocal, with a low neutrophil figure, one may suspect septicopyemia. Meningitis of the convexity can not be separated from that of other areas. Extra-dural and basal meningitis, as well as meningitis complicating cerebral abscess show no characteristic findings aside from neutrophilia; therefore, their presence must be determined by exclusion.

Piney[12] sums up the prognostic value of this laboratory aid as follows:

1. Slight neutrophilia, slight shift to left, and persistence of eosinophiles may be physiological-pregnancy, mild infection.

2. Slight or definite leukocytosis, moderate nuclear shift (not more than four per cent young forms or twelve per cent band forms) and decrease of eosinophiles or lymphocytes—no clear prognosis other than infection.

3. High leukocytosis, great shift to left, decrease or disappearance of eosinophiles, and reduction of lymphocytes—always of grave prognostic import, especially when the percentage of young cells exceeds that of band forms.

4. Progressive fall of total leukocytes, extreme shift to left, total absence of eosinophiles, and well marked lymphopenia, seen in moribund cases.

5. Progressive eosinopenia and increasing leukocytosis—infection to which reaction is occurring. Progressive eosinopenia and falling leukocytosis—infection to which no reaction is occurring.

6. Reappearance of eosinophils and decrease in leukocytes indicates improvement and is accompanied by shift to right.

7. Lymphocytopenia is always a bad sign.

8. Sudden fall in lymphocytes and progressive neutrophilia indicates extension of infection. Prognosis is bad when this occurs with diminution of total leukocyte count and shift to left.

9. Lymphocytosis following neutrophilia and eosinophilia with progression of neutrophils toward normal, both in number and "index," means recovery.

The consensus of opinion of men who have worked with the blood picture is that it is a valuable aid in determining the presence of intra-cranial complications but that it is of very little value in differential diagnosis.

Blood Sedimentation

The blood sedimentation test is a study of the settling power of red cells and is a general biologic reaction akin to fever. It has occupied the attention of investigators since the time of Galen—who called the buff-colored stratum on the surface of a clot "Crusta Phlogistica." John Hunter, in 1791, and numerous other writers made observations concerning it. It dropped into disuse until Robin Fahraeus,[13] in 1917, working in the University of Kiel, rediscovered the phenomenon and reported that it might be used in the diagnosis of pregnancy, acute infection, and malignant tumors. Since that time literature has abounded in reports of attempts made to apply it to practically every field of medicine and surgery. It is in no sense of the word a specific reaction or diagnostic test of any given condition but is an indicator of the presence and, more especially, of the progress of any condition which is attended by cell disintegration and destruction. In any such condition the sedimentation time of the red cells is much shortened.

An explanation of the mechanism of blood sedimentation is outside the scope of this paper. Guida[14] mentions among the theories which have been advanced for its explanation the following: that it is due to the constitution of blood plasma, the constitution of erythrocytes, the presence of megaloblasts, the presence of cholesterin and lechithin in the blood, and the more generally accepted theory that it is caused by modification of the colloidal state of the serum, that is, the altered relation between globulin and albumin.

A study of the literature reveals that the many technics described are all modifications of one or other of two original methods which depend on measuring the distance through which the head of the column of red cells falls in a given time, known as the distance method, originated by Fahræus and modified by Westergren so that it often goes by his name, or in measuring the time required for the head of the column of red cells to settle through a given distance, known as the time method, or Linzenmeier's method.

Westergren uses pipette tubes 300 mm. high and 2.5 mm. in diameter, with a mark 200 mm. from the bottom of the tube. The tube is filled up to the 200 mm. mark with a mixture of one part sodium citrate, 3.8 per cent, and four parts blood, and the

height of the supernatant column of serum read at the end of one, two and twenty-four hours. The normal after one hour, for men, is about 3 mm. and for women from five to 10 millimeters.

Linzenmeier uses small tubes five mm. in diameter and 6.5 cm. high and with five marks, one at the level of the height of a column of one cc. of water and the others at six, twelve, eighteen and twenty-four mm. below this. The tube is filled up to the cc. mark with the mixture described above and the time required for the head of the column to reach the 18 mm. line is determined. For normal men this is about 1200 to 1400 minutes, for women 800 to 1,000 minutes. In menstruation the time is shortened, often to 600 minutes. Anything below 200 minutes is considered definitely pathological.

Cutler[15] has described a graphic method which combines both time and distance factors and feels that this is important in that the rate of sedimentation varies in different stages of the process.

The test has been applied in many conditions, notably in gynecology and tuberculosis, in an effort to differentiate between acute and chronic infections, benign and malignant tumors, aseptic and septic abdominal lesions, non-hemorrhagic and hemorrhagic diseases. It has not proved a specific diagnostic test in the hands of most men but it has been found to be a valuable indicator of the presence of any process, toxic or septic, which is breaking down cellular tissue. Hence, sedimentation time is apt to be decreased in pregnancy, sepsis, malignant tumors and hemorrhage. Workers in tuberculosis have found it of great value and some of them have reported that serial sedimentation tests give more accurate information than the pulse and temperature curve, weight curve, or physical signs and symptoms. The test has so far found but a limited use in otorhinolaryngology.

Weiss[16] found sedimentation normal in chronic otitis media, chronic mastoiditis with cholesteotoma and labyrinth symptoms, and in furunculosis of the canal. Acute mastoiditis and acute otitis media gave decreased sedimentation time. He feels that it can play but a minor role in otologic diagnosis, especially in differentation of intra-cranial complications, but that it may have some value as a guide to the efficacy of treatment and for prognosis.

Bertog[17] found decreased sedimentation time in acute infections of the middle ear; when used serially it is of value in

doubtful cases in differentiating between surgical and non-surgical cases.

Guida,[14] using the technic of Westergren, investigated a series of otological cases; he finds that in acute infections of the middle ear with or without mastoiditis, sedimentation is increased. In acute suppurative otitis media with mastoiditis, sedimentation reaches a high degree, which may vary from a normal of twenty mm. to a maximum of 109; when there is but little involvement of the mastoid sedimentation is less. In suppurative otitis media without mastoiditis, sedimentation is less, varying from ten to thirty mm. the first hour. In bilateral, purulent otitis media with mastoiditis, sedimentation is increased, reaching 119 mm. in the first hour, while in the bilateral form without mastoiditis sedimentation is definitely less. In acute suppurations with increased sedimentation, operation is usually necessary; in other cases with bilateral disease and severe pain in the mastoid, when sedimentation is normal, recovery may occur in a few days. In cases of acute suppurative otitis media, convalescent, sedimentation is diminished. He concludes that in mild aural disease sedimentation is low and that when the disease is severe, requiring surgical intervention, sedimentation is increased. He finds very little differential value in the test.

Blood Dyscrasias

Numerous diseases have their first manifestation in the ear, nose or throat; the lesion may be an angina, adenitis, polyposis, gingivitis, or membraneous or ulcerative lesion in the oropharynx. This is especially true of diseases of the blood and blood forming organs, and it is in this class of cases that the differential blood count becomes of great importance in establishing an early diagnosis. The tonsillar swelling of leukemia, the gingivitis of hemophilia, the adenitis of Hodgkin's disease and the glossitis of pernicious anemia, acute leukemia, hemorrhagic purpura, arsenical poisoning, cachexia, and severe sepsis are well known. These cases may come early to the otolaryngologist and he should realize the value of blood studies in their recognition. If he is wise he will enlist the aid of the internist in the exact diagnosis but he should himself always bear in mind the possibility of meeting them.

Agranulocytic angina and infectious mononucleosis are two diseases which are usually ushered in by malaise and angina and which, therefore, may be seen early by the otolaryngologist. They

emphasize the importance of differential blood counts in obscure or undiagnosed lesions of the nose and throat, but it should be remembered that they are not the only diseases so announcing themselves.

The first case of agranulocytic angina, reported by Werner Schultz in 1922, has been followed in increasing numbers by others, most of them coming from Germany and Austria, with scattering reports from England, America and Italy. The disease is one of unknown etiology, some holding that it is a distinct clinical entity in which the unknown agent attacks the granulocytic elements of the bone marrow, others maintaining that it is a rare reaction to severe sepsis. It may appear at any age and in either sex, but is seen most often in middle aged females. It may or may not be preceded by other diseases. It runs the course of severe sepsis with increasing toxemia and terminates fatally in about 90 per cent of the cases, pneumonia being the most frequent terminal complication. The angina may be present from the beginning or appear later and may vary from the lesions of ordinary, acute, follicular tonsillitis to those of severe ulceration and necrosis, strongly suggesting diphtheria. These lesions may be confined to the tonsil or may be seen on the gums, tongue, jaws, and genitalia, and have been found at postmortem throughout the gastro-intestinal tract. Jaundice is present in about half the cases; hemorrhagic diathesis is found in less than 20 per cent. Gross bleeding is rare, but petechiæ of the gums and mucous membranes have been seen. Cases without tonsillar involvement offer the best prognosis.

Postmortem has shown no special pathology in heart, lungs, kidney, liver or spleen, the findings in these organs being those of acute sepsis, e. g., cloudy swelling and fatty degeneration. The only distinctive pathology is that of the bone marrow, the tissue often being poor or lacking in granulocytic cells, while the red blood cell forming tissue is normal, (so-called "red" marrow). Most of the cells present are lymphoid in nature. Reticulo-endothelial cells are increased. Friedmann[18] observed fatty degeneration of the bone marrow in three cases. Section shows but few granular cells in the tissue around the ulcers, this being taken as evidence of the low resistance of the patient. Vincent's spirochete and fusiform bacillus, bacillus pycocyaneous, pneumococcus and streptococcus have been recovered from the mouth lesions. Blood cultures are usually negative, although streptococcus, pneu-

mococcus, staphylococcus, streptococcus viridans and bacillus pyocyaneous have been found. The opinion has been expressed that such findings should not be taken as proof of the specific nature of the disease, inasmuch as one may get positive blood cultures in the terminal stages of various diseases, notably lymphatic leukemia.

Diagnosis is made from the differential blood count. This shows an absolute diminution in the number of both granular and lymphocytic cells, most marked in the former, so that a relative lymphocytosis exists. The total white count may vary from 200 to 4,600, and the granular cells may be absent or so scarce that monocytes, mast cells, and eosinophils can not be counted. Red cells are normal, or slightly diminished, blood platelets normal or above normal, and hemoglobin normal.

No adequate therapy has been found. Transfusion, x-ray, diphtheria antitoxin, and arsphenamine, have been used without marked benefit.

A complete biblography may be found in the extensive review of Rose and Houser.[19]

Several diseases are characterized by lymphocytosis, among them being acute lymphatic leukemia, various infections such as whooping cough, and a third one, infectious mononucleosis, which is very apt to come to the laryngologist because angina is an early and constant symptom. Its exact identity has not been determined. Filatow[20] regarded it as an idiopathic cervical adenitis; it has been considered by many writers as identical with the so-called glandular fever first described by Pfeiffer.[21] It was established as a clinical entity in this country by the papers of Sprunt and Evans,[22] and Longcope.[23] Downey and McKinlay[24] have called it acute lymphadenosis, and also regard it as a clinical entity. It is essentially a disease of young adults, most often males, is always accompanied by angina, and presents in the blood a cell which serves to differentiate it from the other lymphocytoses.

There is no known predisposing cause or etiology. It is announced by the symptoms of any acute infection, e. g., headache, fever, chill, sore throat, and malaise, usually mild. The angina is constant and early. Physical examination shows various types of throat lesion. There may be only an intense injection of the fauces, or there may be scattered patches of exudate or actual ulcerative lesions of the tonsils. There is present a general ade-

nopathy, most marked in the upper anterior cervical area, but involving also the axillary, epitrochlear, and inguinal glands. Splenomegaly is seen but no liver enlargement. Hemorrhages are rare, although petechiæ occur in the buccal mucosa. Purpuric rash has been reported. The affection is always a mild one and terminates in recovery, usually in two or three weeks.

The differential diagnosis must consider especially acute lymphatic leukemia and infectious lymphocytosis, and is made on the differential blood smear. There is present a moderate leukocytosis, the count ranging from 11,000 to 26,000 and a marked lymphocytosis, the percentage of these cells varying from 57 to 92 per cent. The red cells and hemoglobin are normal or at most show only a moderate secondary anemia.

Whereas most types of lymphocytosis show an immature or lymphoblastic type of cell infectious mononucleosis shows a mature, fully functioning, but abnormal lymphocyte, which, according to Downey, is so characteristic that its presence makes the diagnosis.

The blood differential must rule out especially acute lymphatic leukemia. Early clinical conditions may be very similar but the good outlook in one and the fatal prognosis in the other makes an early differentiation imperative. Acute lymphatic leukemia is characterized by a higher white count, often 50,000 by the time angina occurs, and the cell present is an immature or lymphoblastic cell instead of the mature but abnormal cell of infectious mononucleosis. There is present also a marked anemia, not to mention clinical signs which render recognition possible.

Downey and McKinlay[24] have done the most exhaustive work in the cytology of the disease and Cady (loc. cit.) gives an excellent bibliography.

Bacteriology of the Blood

The bacteriology of the blood has long been studied in general medicine, and septicemia complicating systemic disease, such as typhoid fever, severe pneumonia and terminal erysipelatous infection has long been recognized. The importance of blood cultures in the study of infections of otitic origin was called to the attention of the profession about 1906 by a brief report by Libman[25] to the American Otological Society. This aroused great interest and was followed by much work, until the procedure was placed on a definite basis and given a recognized position among the

diagnostic procedures in otology. A study of the papers which appeared at that time and in the immediately succeeding years gives us most of the information which we now possess concerning the value of this procedure. Particularly important are those of Libman and Celler, Gruening, Sondern, Page, Leutert, Oppenheimer and Hayes, all of which are quoted by Libman[26] in his exhaustive article in the Transactions Ninth. Internat. Otological Congress, Boston, 1912.

As a result of the work which was done it became apparent that a bacteremia may be demonstrated in a certain percentage of aural complications and that this bacteremia is most often seen in sinus thrombosis and meningitis. The question was raised whether or not a bacteremia was present only in complicated otitic infections or whether this finding might also be obtained in uncomplicated cases of acute, suppurative otitis media and mastoiditis. Most of the workers obtained positive blood cultures only in cases of mastoiditis complicated by sinus thrombosis or meningitis. Duel and Wright,[27] however, obtained positive blood cultures in cases of uncomplicated mastoiditis which presented no sign of sinus thrombosis but subsequent workers were unable to duplicate their results.

Cases are frequently reported in which clinical sepsis and positive blood cultures found in aural infections clear up after jugular surgery without the demonstration of a thrombus and the explanation has been advanced that the bacteremia may be caused by thrombophlebitis of small tributary radicals such as the minute veins in the mastoid septa or by phlebitis of a major vessel with erosion of the intima but without actual thrombosis. Kerrison[28] describes an infectious sinus phlebitis without demonstrable clot, with erosion of the intima, and with periodic invasion of the blood stream. Crane[29] states that "it is obvious that we have a goodly number of cases of otitic sepsis due to invasion of the blood stream through channels other than the sigmoid sinus and that we have, often enough, a thrombophlebitis of the smaller veins in the temporal bone resulting in a bacteremia without gross evidence in the large sinus." Eagleton[30] states that "thrombophlebitis of small venous radicals within the cranium is not associated with positive blood cultures; and although cases of positive streptococci blood cultures from thrombophlebitis of small venous radicals in other parts of the body are reported, in infection from the ear a positive blood culture should

be regarded as diagnostic of phlebitis of a large venous sinus." The difficulty in such cases is to rule out a small mural or bulbar thrombus, undetected at operation or autopsy, and the remarks of Libman[26] in 1912 are still pertinent: "The general conclusion one can draw from the literature on clinical and experimental investigations above presented is that while there seems to be a certain amount of evidence that general infections may occur without involvement of the sinus such an occurrence is so infrequent that it plays no role in actual practice and that one has no way of knowing that a given case is the exceptional one in which a thrombosis is not present."

Blood culture has a secure place in otology, even though writers regard it somewhat differently. Lillie[31] says that "blood culture is far more important than a leukocyte or differential count." Kopetzky[2] on the other hand, feels that too much reliance has been placed on blood cultures, that it is a mistake to wait for them, and that more attention should be paid to other phases of the blood picture. Eagleton recommends daily blood cultures, saying that while one or a series of negative reports means nothing, one positive report means everything and Dench[32] states that a "positive blood culture in a case of suppurative otitis with symptoms of sinus thrombosis renders the diagnosis absolutely certain."

Blood cultures may be positive in diseases such as erysipelas, angina, endocarditis, pneumonia or typhoid fever. The first three are most apt to complicate an ear lesion. After efficient blood vessel surgery continuation of a positive blood culture means either that the organisms are multiplying in the blood or that an endocarditis is present.

The consensus of opinion is that a positive blood culture, all other causes being excluded, means thrombophlebitis of a major vessel, and is to be taken as an indication for isolating the ear from the general venous circulation.

Libman[26] gives very concisely the causes for a negative blood culture in the presence of thrombosis. The organisms may be too few in the specimen taken, improper or incomplete cultural methods may be used (both ærobic and anærobic organisms should be sought), the cultures may be made too soon, or they may be made after the discharge of organisms from the local focus has stopped, the bacteremia may be intermittent and the culture made at the improper time, the blood may be so bactericidal that the organ-

isms do not live for any length of time, the ends of the clot may be sterile, the thrombus may be infected but may completely block the vessel, or the thrombus may be sterile.

A consideration of these facts explains the statement that one or a series of negative blood cultures does not rule out sinus thrombosis, and that in the presence of aural sepsis it is not wise to wait for a positive culture before resorting to surgery.

Septicemia complicating rhinological conditions is not common although Libman[26] states that positive blood cultures may be obtained in cases of severe accessory sinus disease and nasal infection. The bacteremia which complicates infections of the upper lip and vestibule of the nose are well known, as is also the cavernous sinus thrombosis which not frequently ensues in such a situation. One finds an occasional case report of bacteremia from organisms commonly found in the nasopharynx, such as micrococcus pharyngitidis siccæ.[33] Such findings, however, are infrequent when compared with those obtained in aural sepsis.

Early and transient bacteremia offers the only explanation for the sequelæ of acute angina. Tanaka and Crowe[34] state that "in ordinary chronic tonsillitis the lining epithelium of the crypt is replaced with scar tissue. Occasionally definite ulcers are found. In other cases large numbers of blood vessels have been thrombosed and they afford a path for the entrance of bacteria into the blood stream." Ballenger[35] goes into the question of bacteremia associated with acute hemolytic stereptococcus throat infections and states that "the fact that complications of acute angina are due to blood stream infection is becoming increasingly evident" and mentions among such complications peritonitis, appendicitis, deep wound infections, and acute thyroiditis. He feels that a bacteremia secondary to an acute throat infection is probably a common occurrence and possibly is the usual accompaniment of this type of infection. He also feels that with an acute otitis media or an acute mastoiditis complicating an acute streptococcus angina a positive blood culture of hemolytic streptococcus is not of as great diagnostic import, so far as thrombosis of the lateral sinus is concerned, as are cultures of other pus-producing organisms. Claus[36] reports a series of twenty-eight cases of post-anginal pyemia with hemolytic streptococcus bacteremia in several; various organisms have been isolated from the blood in agranulocytic angina.

Blood Chemistry

Blood chemistry, while of but limited value in diseases of the ear, nose and throat, is frequently necessary, particularly the study of blood sugar in surgical cases. This should be made in all diabetics, especially those coming up for operation, and the patient prepared by careful feeding and the administration of insulin. This, however, should be done under the supervision of an internist. Hemorrhagic mastoiditis is apt to progress rapidly and painlessly in a diabetic (Kopetzky[2]) and early operation may be necessary.

Estimation of blood calcium has been used by Kopetzky and Almour[37] in the study of undifferentiated deafness. These authors studied a group of cases of middle ear deafness in which the diagnosis of otosclerosis was not certain. They found that the greatest number of patients with diminished blood calcium coincided with those having a true otosclerosis, and that altered calcium content was associated oftener with heredity and raised lower limit than with prolonged bone conduction, history of infection or pregnancy. In eleven out of twelve otosclerotics blood calcium was altered. They concluded that altered calcium metabolism is corroborative evidence of otosclerosis in a patient with deafness and heredity, and that when the deafness began with infection or pregnancy and the patient has an elevated lower limit and prolonged bone conduction, either increase or dimunition of calcium content, and particularly the latter, suggests otosclerosis.

Vermes and Brügel[38], in an extensive study in Neumann's Clinic, made the following observations: Blood calcium in operated and non-operated cases of acute suppurative otitis media and acute exacerbations of chronic suppurative otitis media usually shows some increase, which fluctuates, and is dropping at the time when surgical indications are present. Increased calcium content is found after bone trauma, such as mastoidectomy. Non-suppurative lesions of the middle ear show normal findings. Cases coming to operation late are apt to show normal calcium content because the destructive stage has passed and reparative processes are appearing. These findings are explained by the fact that bone inflammation causes increased absorption of calcium. No operative indications can be drawn from the study of blood calcium.

Novak[39] in a study of hyperesthetic rhinitis, found some of the patients to have a hypocalcemia but does not believe this to be a constant or important finding.

Friesner and Rosen[40] report a series of cases in which the calcium content of the pus from lesions in bone and in soft tissue was analyzed, using the method of Kuttner, and Kuttner's colorimeter. In general, they found that the pus showed more calcium when there was a destructive bone process, abscess of soft parts showing an average of from 7 to 9 mgms. of calcium per 100 cc. of pus, and disease of the bone showing an average of from 18 to 33 mgs. per 100 cc. They found the method a helpful guide but could derive no operative indications from it.

Bleeding and Coagulation Time

Bleeding and coagulation time are of interest on account of their relation to surgical procedures; their value has been questioned and reports indicate a wide divergence of practice in their application.

Bleeding time is the time during which bleeding occurs from a stab incision made with a small lancet or needle, either in the lobe of the ear, or the finger. Osler[41] states that it makes no difference whether the cut is short or long or whether, within certain limits, it is superficial or deep. Dukes' method is widely used; the incision is made and a drop taken up on blotting paper at regular intervals—usually 30 seconds—until bleeding stops. If this occurs within five or ten minutes one may assume slight prolongation of bleeding time; when the twentieth drop is one-half the size of the first there is moderate prolongation. If the twentieth drop is as large as the first there is great prolongation. In a questionable case the test should always be made at more than one place in the skin, and at different times, on account of the variation which the case may present.

Bleeding time does not necessarily parallel coagulation time; it depends on numerous factors, such as the presence of tissue juice, (thromboplastin), elasticity of the skin, sclerosis of the vessels and the mechanical and chemical action of blood platelets. Hemorrhage is controlled by the formation of platelet thrombi in the injured capillaries.[42] When the platelets are reduced, as in purpura hemorrhagica, bleeding time is prolonged. This is explained by the fact that the clot keeps the tissue juice

from reaching the open vessel and by a peculiarity of the platelets which prevents their normal agglutination and disintegration. Normal bleeding time is from one to three minutes but may be as long as eight. It is prolonged in severe anemias, when the fibrinogen content is low (chloroform or phosphorus poisoning) and in destructive bone disease. In thrombopenic purpura (purpura hemorrhagica) it is greatly increased. In all other purpuras it is normal. Some writers have found it normal in hemophilia, others prolonged, but rarely as long as in purpura hemorrhagica, being usually less than eight minutes. Duke found it normal in jaundice.

Coagulation time is the time required for the blood to clot outside the vessel; it is influenced by the temperature of the container, size of the drop, smoothness and cleanness of the instrument, and the method which has been used in withdrawing the blood. Blood taken from a puncture wound will contain some tissue juice and will have a shorter coagulation time than blood drawn by venipuncture. Howell[43] gives coagulation time as twenty minutes (thirty to forty by venipuncture) and Lee and White[44] as ten minutes; their methods with venipuncture are widely used.

Coagulation time is important only when it shows marked prolongation as in hemophilia; Osler states that such variation is the only constant pathological sign of this disease. Slow coagulation is due to delay in the formation of thrombin, not to the action of thrombin or fibrinogen. In any case in which there is a question of hemophilia venipuncture should be used; by this method clotting time may vary from four to forty minutes; when obtained from an incised wound it may vary from five to nine minutes. Coagulation time is prolonged in melena neonatorum, obstructive jaundice, some of the anemias, leukemia, and some of the severe infections such as pneumonia. It would appear that bleeding time is of greatest value in thrombopenic purpura, (purpura hemorrhagica) and possibly in hemophilia, although it may be normal in the latter condition, and that coagulation time is of particular value in true hemophilia.

The relation of coagulation time to post-operative hemorrhage has prompted several recent reports. Kleinert[45] found routine coagulation time of little value and thought there was no particular relation between prolonged coagulation time and post-operative hemorrhage. Other things, such as the age of the

patient, blood pressure, sclerosis of the vessels, and personal and family history are of more importance in warning the surgeon of hemorrhage. McKinney[46] thinks that the test is of no special value but should be done as a matter of protection in case any question arises, and to satisfy the requirement of the patient. Many of them know that such a test is frequently done and do not feel that all precautions are being taken unless it is made. The normal time varies from one to eight minutes; the test would be more valuable if we had a more reliable technic. Hunt[47] found from a questionnaire that the capillary tube method is the one most commonly used in the determination of coagulation time, that the test has but little bearing on post-operative hemorrhage, that it is not necessary if a proper history has been taken and a careful physical examination made, but that in all doubtful cases both clotting and bleeding time should be done. His report would indicate a wide variation in this practice, many surgeons always using it, others never using it, and others using it in selected cases. It appears from a study of all reports that coagulation time under fifteen minutes has no particular relation to post-operative hemorrhage and that in cases where these tests show the most variation, bleeding time in purpura hemorrhagica, and coagulation time in hemophilia, the diagnosis can be equally well established by physical examination and history.

Wassermann

The following statements relative to the practical value of the Wassermann reaction may be made:[48]

1. It is of special value in extra-genital sores and atypical lesions of all stages.

2. A negative reaction is of less value than a positive.

3. In early latent syphilis after the chancre has healed and before secondary lesions have appeared the Wassermann is the only diagnostic test.

4. It is often the only diagnostic aid in late and tertiary syphilis.

5. It may be negative in the blood serum and positive in the spinal fluid in cases of cerebrospinal syphilis.

6. A persistently positive reaction means the presence of living spirochetes in the body.

Syphilis occurs not infrequently in otolaryngology and the Wassermann reaction should be used freely in attempting to

clear up the diagnosis of obscure lesions. Fournier[49] found 850 extra-genital chancres in 10,000 cases of primary syphilis, and of these 75 per cent were in the region of the head, mostly about the mouth and throat. Fifty per cent of all extra-genital chancres were on the lips.

St. Clair-Thompson[50] has this to say about the occurrence of syphilis in the nose and throat. "Primary syphilis of the nose has been diagnosed but is rare. Secondary syphilis of the nose is almost unknown, being most commonly seen as the "snuffles" in infants suffering with hereditary lues. It may give a very persistent nasal catarrah in adults, which resists all treatment and does not respond to cocain. Mucous patches in the nose are rare."

Tertiary syphilis of the nose is found in the form of gumma, which is the foundation of all manifestations of that stage which appear in the nasal cavity, such as typical gumma, with or without ulceration, perichondritis, with or without necrosis, and certain types of atrophic rhinitis. The Wassermann should be used in cases of atrophic rhinitis, perforation of the vomer, ulcerative processes, sequestra, or perichondritis. It is a good preoperative measure in certain patients who are to have nasal surgery, particularly submucous resection, especially when there is any suggestion of syphilis. Such surgery should not be done if there is any evidence of an active process in the nose. Occasionally a Wassermann test will reveal the cause of delayed healing after submucous resection when the nose fills up with a gummy, adhesive process. Such cases may clear promptly under specific treatment.

Secondary syphilis of the larynx manifests itself usually in the form of erythema or mucous patches. Tertiary syphilis of the larynx takes the form of gummatous infiltration with or without ulceration. Due to the fact that syphilis of the larynx may occur with or without tuberculosis and carcinoma the Wassermann reaction in a suspected lesion of the larynx does not have an absolutely conclusive significance. A positive Wassermann in the presence of such a lesion means, as before stated, that the patient is syphilitic. It does not necessarily mean that the lesion under consideration is luetic. St. Clair-Thompson, in summing up the situation says: "The Wassermann reaction is helpful but not conclusive."

In otology the indications for the Wassermann test are perhaps more limited than in rhinology and laryngology. Rarely, ulcerative lesions of the canal or auricle are leutic[51]. The role of syphilis in diseases of the internal ear is well known; suffice it to say that any case of sudden profound deafness, with marked diminution of bone conduction and depression of the upper limits of hearing, or any case of sudden, severe tinnitus and vertigo should have a Wassermann. It should be done in cases of congenital deafness.

Cerebrospinal Fluid

The normal pressure of the cerebrospinal fluid is always the same as the general intra-cranial pressure as measured in the torcular herophili and varies from 60 to 150 mm. of cerebrospinal fluid, being greater in the ventricles than in the subarachnoid space. Increased pressure is seen in cases of cerebral tumor, hemorrhage, hydrocephalus, meningitis, and encephalitis; it may reach 500 or 1,000 mm. of fluid. Low readings are found when the fluid in the spinal column is not in full communication with that within the cranium, as in spinal tumor, Potts' disease, spinal and cerebrospinal meningitis with adhesions, or in spinal compression.

Changes in appearance may be due to the presence of color or turbidity, neither of which is ever present in a normal fluid. Coloring varies from a faint, pale yellow to a deep chrome. It is seen in meningitis; a yellow color is constantly present in subarachnoid hemorrhage.

Turbidity may vary from a slight trace to that of thick pus. When only a trace is present one must rule out chemical coagulants, such as carbolic acid or alcohol, which may have been present in the needle, or traces of blood. Turbidity is caused by the presence of cells, organisms, or fibrin web. Neutrophils are more likely to cause turbidity than lymphocytes but the clouding depends on the death and degeneration of cells rather than on their nature. A count of over 300 neutrophils usually gives an obvious turbidity, while one may have over 500 lymphocytes with a clear fluid. Cloudiness is no indication of the severity of the infection.

The fibrin coagulum, which is the most delicate test for fibrinogen, is often missed because the fluid is shaken up in transport to the laboratory; when a fluid is being examined a

small portion should be saved with the object of watching for the formation of the web. This may vary from a fine, delicate skein to solidification of the whole tube and is particularly apt to be found in tuberculosis and syphilis, especially with highly albuminous fluids. Any increase in protein above 100 mgms. per 100 cc. may be accompanied by fibrin coagulum; it may not form, however, owing to the absence of the fibrin ferment, even in the presence of considerable fibrinogen. This may be supplied by the addition of a drop of fresh blood or serum, when coagulation will occur.

Neutrophils are not found in normal fluid but even a trace of blood will cause them to appear. They are seen in pathological conditions, especially acute infections and are apt to dominate the picture in acute coccal meningitis and cerebral abscess; they may constitute fifty per cent of the cells in tuberculous meningitis and poliomyelitis. Lymphocytes are found particularly in syphilitic conditions and out-number the other cells except in acute epidemic, coccal meningitis and brain abscess. Large mononuclear cells (endothelial cells) are always present in pathological fluid but never constitute more than ten per cent of all cells. Eosinophils are present in small numbers in acute meningitis. Plasma cells are characteristic of chronic diseases of the cerebrospinal system. Compound granular corpuscles are seen in cerebrospinal lues, and tuberculous meningitis; macrophage cells occur in subarachnoid hemorrhage and tumor, but have no special pathological significance.

The normal fluid contains very few cells. Nonne gives 5 per cubic mm. as normal. Other writers say that three is the average and that anything above four is pathological. Greenfield and Carmichael[52] classify pleocytosis as follows: 5 to 10 per cubic mm.—slight, 10 to 50—moderate, 50 to 250—severe, and above 250, extreme.

These authors divide the cytological changes into three types, the first, in which are found lymphocytes and endothelial cells of the so-called mononuclear type, the second, in which is found the mononuclear cell with some pathological cell, such as the neutrophil, and the third, which is essentially neutrophilic; they point out that with slight reaction the lymphocyte is the cell most apt to be found, with severe reaction (50 to 250 cells) both the mononuclear and the neutrophil, and in the very severe type the neutrophil.

The estimation of protein has been of value in measuring inflammatory changes in the meninges. The two proteins most commonly found are globulin and albumin, the latter present in the greater amount; there is no known condition in which globulin exceeds albumin. A mild or moderate increase in protein is the commonest chemical change in the cerebrospinal fluid; a great increase is rare. The normal content is from 25 to 100 mgm. per 100 cc.; increase up to 500 mgms. is seldom seen; content above 500 mgms., rare. Increased protein is apt to occur as an isolated phenomenon or with altered color and increase of cells, and is characteristic of brain tumor, cord tumor, cord compression, and localized spinal meningitis. Increase of protein with increase of cells is the commonest pathological finding and is seen in all forms of meningitis, acute, subacute and chronic, and in various other lesions, but the protein increase is not always parallel with the cell increase. In general, in meningitis the protein does not rise above 200 mgms. per 100 cc. of fluid; rarely it goes to 750, 2,000, or even 3,000 mgms. Such fluids, with scant or absent cells suggest a meningitis limited to the upper part of the cord and base of the brain.

Glucose is normally present in amounts varying from 45 to 85 mgms. per 100 c.c. The blood contains more sugar than does the cerebrospinal fluid and its sugar content varies more rapidly with physiological and pathological conditions than does that of the spinal fluid. Increased glucose content in spinal fluid is seen as an accompaniment of increased blood sugar in diabetes mellitus and acute nephritis, and may reach 100 mgms. per 100 c.c. in many general diseases. Diminution is a cardinal sign of acute meningitis, whether tuberculous or coccal, but is usually slight in the early stages; in late stages sugar may be absent. This is due to the presence of ferments which result from cellular disintegration, or of organisms which use glucose as food. The variation in glucose is most important in acute meningitis.

Chlorides are normally present in from 720 to 750 mgms. per 100 c.c. of spinal fluid. This figure is so constant that any variation is regarded as pathological. Any increase above 750 mgms. is indicative of renal deficiency; figures lower than 720 mgms. are always important and usually pathological. Certain conditions, such as brain tumor and cerebral abscess may give a cytological picture much like acute meningitis which may be ruled out by normal chloride content. A progressive lowering of

the chlorides means invasion of the subarachnoid space; in acute meningitis the figure may drop to 650 mgms. per 100 c.c. In tuberculous meningitis particularly, the diagnostic value of the chloride content is second only to that of the presence of tubercle bacilli. The chloride in cerebrospinal fluid never falls below the blood chloride; progressive lowering or a single low reading is a bad sign.

Increased meningeal permeability appears in pathological conditions and has been made use of as a diagnostic aid. Sodium nitrate particularly has been so used. One gram by mouth for each 30 kilos of body weight should normally show from 1 to 1.5 mgms. per 100 c.c. of spinal fluid in three hours. In meningitis this may be increased to 5.5 mgms. and in tuberculous meningitis to 8.5 mgms.

Kafka in 1912 found that uranin (sodium fluorescein) by mouth in 6 to 8 gram doses appeared in the spinal fluid in 30 per cent of non-syphilitic and 100 per cent of paralytic cases. In the majority maximum elimination was at the end of three hours but traces appeared anywhere from one to seventeen hours.

Most writers describe a serous meningitis, a suppurative meningitis, and a condition known variously as meningism or meningismus. Suppurative meningitis is divided into circumscribed and diffuse and the same classification is occasionally found in the descriptions of serous meningitis.

By serous meningitis is meant an inflammatory condition of the arachnoid or pia, usually secondary to some lesion, such as extra-dural abscess, in which these membranes, though congested and edematous, have escaped actual invasion, there being present secondary edema but no pus or bacteria (Kerrison[28]). Politzer[51] describes a serous infiltration of the pia mater caused by inflamed meninges and accompanied by an increase in the amount of cerebrospinal fluid. Greenfield and Carmichael[52] state that the term includes a great variety of mild infections of the meninges due to the presence of avirulent organisms in the subarachnoid space, or a more virulent organism in the layers of the dura or the subdural space. Kerrison states that in purely serous meningitis organisms and pus cells are not usually found but Yerger,[53] in a recent study, found organisms in 30 per cent of the cases with a cell count of 250 or less. In this type of meningeal involvement the fluid is usually clear but on occasion may be

cloudy; there is usually no color and the pressure is increased. The count is usually low (below 250 per cubic mm.) and the cells of the lymphocytic type, although the neutrophil may predominate. Globulin is increased; sugar is diminished or absent.

By circumscribed, suppurative meningitis is meant a process limited by marginal adhesions which prevent a diffuse meningitis but do not keep organisms from reaching the spinal canal. The fluid may yield both cells and organisms and "the presence of pus and bacteria in the fluid may constitute no evidence of the existence of what may properly be called a diffuse, purulent meningitis (Kerrison). Politzer describes a leptomeningitis circumscripta, which is a walled-off collection of pus between dura and pia, and Yerger mentions a circumscribed, suppurative meningitis which includes the cases of so-called protective or sympathetic meningitis. The fluid is turbid or frankly purulent and may present a faint greenish yellow or yellow color; the pressure is increased. The cells vary from 250 to 5,000 (Yerger[53]). He makes the cell count one of the criteria for differentation of circumscribed from diffuse meningitis, counts under 5,000 usually indicating a circumscribed process and those above 5,000 a diffuse process. The cell predominating is the neutrophil. The total protein may vary from 100 to 200 mgms. per 100 cc. Globulin is increased. Sugar is reduced or absent and the chlorides may be reduced as low as 600 to 650 mgms. per 100 c.c.

Diffuse, suppurative meningitis is a general invasion of the subarachnoid space by the infectious process. Some authors do not divide such a process into circumscribed and diffuse, but such a classification is usually found. The basis of classification depends on the cell count and the severity of the bacterial invasion, low counts pointing toward a circumscribed process and high counts indicating a diffuse process. Yerger places cases with a cell count of 5,000 or under in the circumscribed class and those with 5,000 or more in the diffuse, and states that the presence of bacteria in cerebrospinal fluid is the best clinical evidence of a diffuse meningitis. Kerrison disagrees and says that the presence of pus and organisms does not necessarily mean a diffuse, purulent process. Greenfield and Carmichael state that a large number of organisms in smear and culture indicates an impending diffuse meningitis and that diminution of sodium chloride to 680 mgms. in a turbid fluid has the same significance. Cell counts up to 1,000, protein increase up to 100 mgms. and

absence of glucose do not necessarily mean diffuse meningitis as long as the chlorides remain normal. A fluid containing only a few avirulent organisms does not necessarily mean a diffuse process. Most reported cures of meningitis are those classified as circumscribed, with or without organisms, and with low cell counts, often predominately lymphocytic.

The condition known as meningismus usually presents a fluid normal except for increased pressure.

The fluid in brain abscess may be perfectly normal or it may present the picture of a circumscribed meningitis with a sterile fluid which may be either serous or purulent. The count may be very low, under 10, and predominately mononuclear; Yerger found it to be under 500 in 95 per cent of his cases. Eagleton points out that the count is apt to be high in the early stages, dropping later as the process becomes walled off, when it may be as low as 250. The pressure is increased, globulin may be present and sugar diminished or absent. The spinal fluid findings are influenced by the proximity of the abscess to the subarachnoid space. Normal fluids may be found, as before stated, if the abscess is deeply seated and well walled off. If it is near the surface the fluid is apt to show the changes characteristic of the complication accompanying the abscess. In localized processes such as extra-dural abscess the picture is apt to be that of serous meningitis or circumscribed suppurative meningitis; if true diffuse meningitis is present then the fluid findings will be those of the latter process.

It is important that the spinal fluid be studied in conjunction with other signs and symptoms. A brain abscess may have a perfectly normal fluid or it may present the picture of a circumscribed meningitis. A serous meningitis may or may not have organisms as may also diffuse, suppurative meningitis. The cell count, while giving us some information, does not definitely classify the spinal fluid. In general it may be said that serous meningitis is apt to have a clear fluid under pressure with a low count, predominately lymphocytic and that suppurative meningitis is more apt to have a turbid fluid with a high count, essentially neutrophilic, and with a greater number of organisms present.

Lumbar puncture should be done daily in cases of suspected intra-cranial involvement and in the post-operative care of brain abscess in which it gives us our most exact knowledge of the

changes going on in and about the lesion. It is well to do lumbar puncture before operation in chronic, suppurative otitis media; it may reveal the presence of an unsuspected complication.

In 1925 Dowman[54] described compression of the jugular veins in the diagnosis of thrombosis of the lateral sinus; Tobey and Ayer[55] reported in greater detail their experience. The procedure is based on the work of Queckenstedt and depends on variation of pressure of cerebrospinal fluid. Absence of rise of fluid pressure on jugular compression may indicate spinal subarachnoid block, as seen in cord tumor and tumor of the cerebellar fossa, or venous blocking, as seen in sinus thrombosis.

The test is made by doing lumbar puncture, using either a water or mercury manometer, and then gradually compressing first one and then the other internal jugular, noting the variations recorded in the fluid column. In thrombosis the pressure will be increased two or three times normal when the patients jugular is compressed, while pressure over the thrombosed vein causes no rise or a slow rise of from 10 to 20 m.m. This is usually indicative of complete obstruction. Partial obstruction gives less striking results.

The procedure occasionally shows no rise of pressure when obstruction is absent. This is usually attributable to the fact that the pressure is abnormally low or that the jugulars are inaccessible. Low pressure may be corrected by the injection of 10 to 20 c.c. of salt solution when the test may show normal results. Abnormal increase of pressure in the absence of obstruction is usually due to carelessness in making the test. The authors found that a difference of 50 m.m. between the two sides was unusual and one of 100 m.m. was exceptional; with a normal initial pressure (150 m.m.) the rise will seldom be less than 50 m.m. and more likely will be over 100.

Anatomical variations in the veins explain slight rise of pressure sometimes obtained after compression of the thrombosed side. The test must be carried out under exact technic. Death from medullary pressure is possible; acute meningitis precipitated by the procedure was not noted.

Gaillard and Mayoux[56] consider the test an excellent means of diagnosis in block of the subarachnoid space. Not only must the subarachnoid space be free but the venous system must be normal in order to demonstrate normal resistance to jugular compression. Elevation of pressure may transmit itself from the

jugular to the cerebrospinal fluid by anastomosis between the lateral sinus and the internal jugular which are insufficient in most cases to render the test inaccurate. These authors do not feel that compression of the cervical sympathetic plays any part in the result. One case is reported in which the test was positive, although the sinus was normal, and one in which the test was negative in the presence of a thrombosed sinus. In the first instance increased pressure was due to compression of the sinus by a perisinus abscess. Such pathology, however, does not always give a positive reaction. They conclude that the test is of definite value in diagnosis of thrombosis of the lateral sinus and recommend its systematic use, bearing in mind the possible causes of error.

Aubry[57] reported a case of obliterative thrombophlebitis in which the test was negative; this was explained by the slow evolution of the occluding process and by coincident serous meningitis which had persisted for a long time after disappearance of the focus of infection.

Mayoux[58] concludes that Queckenstedt's test is of great interest in the diagnosis of thrombosis of the lateral sinus and should be included in daily practice.

Bacteriology

A great many studies have been made of the bacteriology of acute and chronic infections of the middle ear; the streptococcus hemolyticus, the pneumococcus, the streptococcus mucosus (pneumococcus type III), and the staphylococcus, have been named as the most important, with a long list of other organisms which includes the diphtheroid bacillus, bacillus proteus, bacillus pyocyaneus, and bacillus coli. The exact relative importance of the first group has varied somewhat with different writers but in recent literature the most importance, particularly as regards the severity of the infection and tendency to produce intra-cranial complications, has been attributed to the streptococcus hemolyticus and streptococcus mucosus, with the other organisms playing a minor role.

Bacteriologic studies of acute ear infections are best made at the time of paracentesis or spontaneous rupture, under conditions which as far as possible avoid contamination from the skin of the canal; the organism responsible for the pathology can be recovered in most cases. Contamination soon occurs and the

primary organism may be overgrown by invaders so that cultures taken from an acute otitis media of several days' duration are much less reliable in telling us the organism primarily responsible. This is true also of cultures taken in chronic, suppurative otitis media in which contamination has long existed so that the original infection has been overgrown and perhaps lost. Cultures taken under such conditions have very little value.

One of the best recent studies of the bacteriology of acute otitis media is that of Valentine[59] who followed a series of acute middle ear infections over a considerable period of time and attempted to correlate the changing bacteriologic picture with the varying clinical appearance. She found, as have most other writers, that the original infection is monobacillary; after a few days it becomes mixed due to contamination. Beta hemolytic streptococcus was the probable cause of infection in over 50 per cent of her cases; there was marked correlation between the bacteriology of the throat and ear when the beta hemolytic streptococcus, the green streptococcus, or the pneumococcus was found in the aural secretion. The green streptococcus is found in normal throats. Beta hemolytic streptococcus is easily overgrown by staphylococcus and diphtheroid bacilli, and normal throat inhabitants such as the green streptococcus, and gram negative coccus come to the foreground. This has been mentioned by other writers who have referred to the disappearance of the secondary invaders under treatment and the later reappearance of the primary organism with exacerbation of symptoms. Ninety per cent of the cases showing beta hemolytic streptococcus in the ear showed the same organism in the throat. The pneumococcus was found in much smaller number and the staphylococcus alone in 22 per cent of the acute cases and alone or combined with other organisms in 70 per cent. The question of contamination enters in here very largely. The diphtheroid bacillus was infrequent in primary culture but played a prominent role as secondary invader, being found in 56 per cent of the cases after one to three weeks. Valentine's final conclusion was that the acute cases are due most frequently to the beta hemolytic streptococcus, the staphylococcus, the pneumococcus, and the green streptococcus, and the chronic cases to the staphylococcus, bacillus pyocyaneus, bacillus proteus, bacillus coli and the diphtheroid bacillus. Bacteriology alone does not account for the development of chronicity.

In the study of acute aural infections certain organisms assume peculiar importance. Hemolytic streptococcus is prone to cause a fulminating type of mastoiditis and intra-cranial complications; hence studies on blood agar plates should always be made. The streptococcus mucosus capsulatus, now generally recognized as pneumococcus type III, is another organism which is characterized by its insidious and extensive invasion of bone tissue and subsequent sudden onset of intra-cranial complications. This organism is easily recognized in bacteriologic studies by its bile solubility, and by its capsule, and any ear lesion due to it should be watched very carefully for early operative indications.

There are numerous case reports of diphtheretic otitis media, and Drury[60] has presented a very thorough study of this particular infection. Kerrison states that five per cent of all cases of diphtheria develop some type of acute otitis media; Duel puts the figure at ten. Only a small percentage of these are true primary diphtheria but many of them are due to diphtheria associated with other organisms. A diphtheretic otitis media is occasionally seen in a carrier. The infection occurs through the eustachian tube in cases of pharyngeal diphtheria; Drury feels that the organism probably comes through the blood stream rather than from the pharynx. Otitis media due to a pure infection of diphtheria bacilli is characterized by watery discharge, sodden appearance of the drum, and the possible presence of membrane about the perforation; such findings would suggest the advisability of culturing the discharge on the usual Loffler medium and treatment with anti-toxin.

Tuberculosis of the middle ear is a disease the diagnosis of which may be made by bacteriologic or pathologic methods. The tubercle bacillus is recovered with difficulty from the aural discharge, particularly in the chronic cases, which often show an acid fast bacillus which must be carefully differentiated by proper staining reactions. The organism may be found in the discharge from acute cases. An attempt to establish the diagnosis by bacteriologic methods involves the collection of the secretion and its examination either in smear preparation or by injection into guinea pigs. The material for smear preparation should be treated with antiformin and centrifuged, while that which is to be used for animal inoculation should not, of course, be treated with antiformin. Wingrave[61] reported finding bacilli in either discharge or curettage material in 16 per cent of the cases of

chronic tuberculosis and in 87 per cent of the acute cases. Such studies should be made in cases presenting multiple perforations of the drum, or in long standing cases which have resisted all therapy and in which bacteriologic studies have given no clue.

One should bear in mind the possibility of finding Vincent's spirochete and the bacillus fusiformis in suppurations of the middle ear. Pilot and Pearlmen[62] found these organisms in the foul discharge of chronic, suppurative otitis media, associated usually with streptococcus, pneumococcus, or diphtheroid bacillus; it was not found in the normal ear canal, in acute infections, or in non-fetid discharge. The organism comes through the Eustachian tube.

Otomycosis is another aural infection, the diagnosis of which is established by bacteriologic methods. Chisolm and Sutton[63] feel that it is more frequent than is commonly suspected. The fungi belong to two families, the mucoraceæ and aspergillaceæ, seventeen species of which are pathologic for man; ten of these have been reported in various ear conditions, the most common being the aspergillus fumigatus. These organisms are probably never primary invaders but occur secondarily in other pathological conditions; they are found particularly when maceration of the skin has occurred as a result of trauma or prolonged soaking in water. The mycelium may occur not only in the external canal, but also in the middle ear itself. If the infection remains superficial it may cause only slight itching, a feeling of fullness, and impairment of hearing; if the deeper tissues are invaded there may be intense itching, with swelling of the canal, tenderness, and dull pain. If the middle ear is involved there is rather profuse discharge of mucus. The infection, particularly in the dry cases, can be recognized by the fine brown, yellow or almost white flakes which present in the external canal. The diagnosis is made by finding mycelia or spores in the slides or in cultures in Sabouraud's agar or Raulin's liquid.

Bacteriologic studies of the mastoid at operation indicate the responsible organism in a large percentage of cases and when an intra-cranial complication is present will frequently reveal the organism which is responsible. In acute cases some type of hemolytic streptococcus is usually found. In a series of thirty-five cases 48.3 per cent showed this organism. Such cultures should always be taken whether intra-cranial complication is present or not.

Among nasal conditions which may be diagnosed by bacteriologic methods are Vincent's infection, diphtheria and primary syphilis. Nasal cultures in pharyngeal diphtheria should be a matter of routine in convalescence in order to rule out carriers. Diphtheria will always suggest itself when one sees in the nose a white, adherent membrane, accompanied by profuse, watery discharge and marked nasal occlusion; such membrane may be seen in scarlet fever and severe streptococci infections and it is quite essential to rule out its specific character. The fusiform bacillus and the spirochete of Vincent have been reported in severe nasal lesions, particularly in small children, and smear examination should always be made when there is infection of unknown etiology, particularly if accompanied by profuse, bloody discharge.

Dark field examination for the spirochete pallida should be made in all lesions about the nose when there is any possibility of the condition being syphilitic. Chancre of the septum is a comparatively rare occurrence but occasional cases are reported and demonstration of the specific organism will allow treatment to be started when it is of greatest value.

The bacteriology of the throat, like that of the ear, has been the subject of many elaborate researches, most of which have concerned the hemolytic streptococcus. Pilot and Davis[64] found this organism in the crypts of 95 per cent of excised tonsils, on the surface of 61 per cent of such tonsils, and in 43 per cent of nasopharynges. Bloomfield[65] found in the normal throat a non-hemolytic streptococcus, a gram positive coccus, and the influenza bacillus and in lesser numbers pneumococcus type IV, the diphtheroid bacillus, and the staphylococcus. He found that the beta hemolytic streptococcus is not a normal inhabitant of the throat and that its presence indicates a focus of infection; it was present in 9.5 per cent of the tonsillectomized cases and in 41 per cent of patients with tonsils. It is particularly located in the bottom of the crypts.

It is academic to speak of the diagnosis of diphtheria by bacteriologic methods and yet there are one or two points which might be mentioned. In suspicious cases one should not be satisfied with one negative culture but should continue taking them daily until the case either clears up or the diagnosis is established. Here, as in other laboratory methods, a negative result does not rule out the condition suspected. Cases have been reported in

which anti-toxin was withheld on account of negative cultures only to be given too late after positive cultures had been obtained. The common practice of smearing the swab over the surface of the membrane is not the best way of taking the culture. If possible the membrane should be lifted up and material obtained from its edge, or preferably under surface, where the organisms are most apt to be found. Many bacteriologists will not make an absolute diagnosis of diphtheria from the smear preparation but prefer to wait for an eight or ten hour culture.

Infection with Vincent's spirochete and fusiform bacillus is common and its diagnosis well understood. The organisms are found about the teeth and tonsils in poor hygienic conditions and are frequently responsible for severe gingivitis and tonsillitis. The presence of a few organisms in the smear is not sufficient evidence for concluding that they are causing the pathology present; they should predominate to permit this conclusion.

Pilot and Brams[66] found the fusiform bacillus in 82 per cent of extirpated tonsils and 32 per cent of extirpated adenoids and the spirochete of Vincent in 25 per cent of such tonsils and 2 per cent of such adenoids.

The dark field is important in the diagnosis of suspicious lesions in the mouth and pharynx, particularly the primary sore and the mucous patch; it should always be done when there is any questionable ulcer in these areas.

Actinomycosis is a disease which is peculiarly apt to be found about the head and neck. New and Figi[67] report a series of 157 cases, 107 of which occurred in these areas, and state that the cervico-facial group makes up the majority of these infections. Actinomycosis of the tongue is reported in increasing number and in any obscure lesion of the tongue, glands of the neck, skin or subcutaneous tissue of the cervico-facial region in which malignancy, Hodgkins, or phlegmon is suspected, watch should be kept for the ray fungus and sulphur granules of actinomycocis. These are the macroscopic yellowish white bodies seen in the discharge from actinomycotic cavities and should always be looked for, particularly in freshly opened pockets and deep crypts. Diagnosis is made by crushing a suspected granule between slides and examining for the characteristic mycelia and spores of actinomycosis. The organism is grown culturally but it is not a simple matter. If mixed infection is present the fungus

may not be found in the discharge, but must be looked for in the tissues themselves.

Pathology

Pathologic studies have but a limited use in otology inasmuch as malignant disease of the ear is not common. In a case with polyp or granulation tissue which bleeds freely, recurs promptly, or has any feeling on palpation suggestive of malignancy, biopsy should be done. It may also be done on granulation tissue from chronic suppurative otitis media when the exact etiology is unknown and tuberculosis is suspected; a small bit of tissue may be punched out and examined for tubercle bacilli and giant cells. Wingrave[61] states that chronic tuberculosis is characterized by the finding, in curettage material of giant cells but rarely tubercle bacilli, and that acute tuberculosis more frequently shows tubercle bacilli, and less often giant cells. One must be careful to differentiate the true giant cell of tuberculosis from the large mononuclear cell frequently seen in non-specific bone granulomata.

In rhinology very much the same general indications for biopsy hold. Whenever a case presents in which the tissue has any suggestion of malignancy, either in free bleeding, early recurrence, or in the hard consistency of cancer, pathological sections should be made; frozen sections during operation are occasionally desirable when one is desirous of knowing whether the tissue under immediate consideration is malignant or not.

Three pathological processes occur in the larynx, which are often difficult of differentiation, namely, tuberculosis, syphilis and cancer. The question of whether or not biopsy should be done in suspicious lesions of the larynx has long been the subject of spirited discussion. Some maintain that biopsy should not be practiced because of the danger of causing metastasis, others that it should be done only when all other means of diagnosis have been exhausted and the nature of the lesion is still in doubt, while others maintain that biopsy, being a harmless procedure, should be used freely in every suspicious lesion of the larynx, because there are many cases in which the diagnosis can not be established without its aid. All agree that if tissue is taken it should be done with the least possible trauma, that crushing and bruising manipulations should be avoided, that a sharp punch forceps or cutting instrument should be used, and that the procedure should not be carried out unless permission has been obtained for im-

mediate operation in case malignancy is diagnosed. The question is not settled; the solution apparently depends on the experience of the individual operator and his ability to diagnose malignancy without the microscope.

Actinomycosis is another lesion that may be diagnosed by biopsy, even in the absence of sulphur granules, and the possibility should always be borne in mind in obscure lesions about the head. Jackson[68] has reported a case of blastomycosis of the larynx, in which the diagnosis was made by biopsy, and culture.

The usefulness of biopsy in general can be stated by saying that whenever we have a tumor, granulation, or ulcer of unknown character which is surgically accessible and from which tissue can be removed without hazard to the patient, such study should be made. One criticism of the use of biopsy has been the fact that it is sometimes difficult to be sure the specimen has been taken from the true tumor, and not from harmless granulation. Tissue should be taken from the edge and from the center of the growth; even under such conditions first specimens are occasionally reported negative and later ones positive. The explanation is, of course, that the biopsy missed the tumor the first time and found it the next. A second difficulty is the varying reports made on the same slide. Despite these shortcomings the procedure is well recognized and widely practised.

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DISCUSSION:

DR. FRANK R. SPENCER, Boulder, Colorado: The white blood count gives valuable information in differential diagnosis between acute suppurative otitis media and acute mastoiditis when high. It is of very little or no value in chronic otitis media or chronic mastoiditis. If the count is very low or normal, it is of course of no value except with an overwhelming acute infection. The white blood count helps in the diagnosis of sinus thrombosis as a sudden elevation of the blood count corresponds with the sudden elevation of temperature. At most, when the white blood count is high it means only an infection somewhere in the body. The great importance of blood examination in agranulocytic angina has been emphasized by the majority of those reporting such cases. Internists tell me they used the Arneth or Schilling test, but found this of very little practical value, so it is not used as much as formerly.

The reports in the literature are to the effect that blood cultures are of relatively little value. While I am personally in favor of taking a blood culture, so few are positive that the negative reports are of little or

no value. It is sometimes not to the best interest of the patient to wait for a positive blood culture, just as it is not to the best interest of the patient to wait for a positive culture in suspected diphtheria before giving antitoxin when clinically diphtheria is the most likely diagnosis. We all see very definite cases of sinus thrombosis with a negative blood culture, yet we do not feel justified in waiting for a positive blood culture. A blood culture in meningitis may be positive late in the course of the disease, but it is usually negative early. The essayist has emphasized one very important feature when he says, "The consensus of opinion is that a positive blood culture, all other causes being excluded, means thrombophlebitis of a major vessel, and is to be taken as an indication for isolating the ear from the general venous circulation."

The essayist emphasizes the importance of blood chemistry, particularly in diabetics, and the value of insulin. Testing for sugar tolerance is important. It is also important in tumors of the hypophysis.

We ordinarily test the clotting time of all patients likely to require an operation, and in some the bleeding time is also tested. We have tested the clotting time for 2290 during the past ten years, but by no means have all of these required an operation. This test has been of very little practical value except to impress the patients and the patient's relatives with the fact that every precaution is being taken. Many patients know about the test and are disappointed if it is not made. We failed to discover one case of purpura haemorrhagica before operation, with the result that the patient died ten days after operation in spite of every known method to control his hemorrhages. My chief, Dr. Robert Levy, tells me that he does not pay very much attention to the clotting time, yet I am sure his results are at least as good as they would be if he had the test made routinely.

Doctor Connor does not mention the Hinton test for syphilis. This test has been used extensively at the Massachusetts General Hospital for the past ten years. Their reports indicate that this test is more valuable than the Wassermann. We need constantly to be reminded that any single laboratory test, or any other test for that matter, may be misleading.

Doctor Connor mentions jugular compression for the diagnosis of lateral sinus thrombosis as reported by Dowman in 1925. Crowe of Johns Hopkins described this test about 1910, so that it is often known as Crowe's test.

One of the most useful methods of securing pus from the middle ear in suspected tuberculous otitis media is by means of a small aspirator. I am passing around several pictures to show this. The external auditory canal and middle ear must be carefully cleansed with cotton wound applicators and then wiped out with alcohol to avoid obtaining secondary organisms. Pus aspirated from the granulations of the middle ear will often show tubercule bacilli. If not, an animal inoculation may be required. However, animal inoculation is not entirely satisfactory because the animal may die in a very short time from the secondary organisms. Haskin and Dryer advocate the use of Petroff's media, which was the original media used by Dwyer at the Manhattan Eye, Ear, Nose and Throat Hospital. They advise that this be used with Miller's later media made from the spleen and lymphatic glands of animals. It is easy to get

a growth of tubercle bacilli in from ten to twenty days. They also advise the use of antiformin before staining from tubercle bacilli, but not if the pus is to be injected into an animal.

Dr. Gordon Berry, Worcester, Mass.: Yesterday Dr. Eagleton told us to take daily visual fields in our brain abscess cases, but he warned us against attempting this task ourselves, or it would not be done. Somewhat the same advice could be given about our carrying out careful laboratory tests. This is a most important phase of our work, but many of us are incompetent to conduct it, or have not the time. There are those who can, however. In proportion to our seeking this laboratory aid we will develop our usefulness. Some of our number are themselves expert pathologists. Ten years from now laboratory workers will be so indispensable to our best work that we will wonder how we ever stumbled through successfully without them. We owe much to Dr. Connor for bringing this data before us. Let us illustrate briefly its present clinical value.

A blood picture is a help in differentiating lymphoid tumors. This was shown in a young man of about 23 years. Last Christmas a lateral neck swelling was incised and a staphylococcus aureus recovered. The wound healed but the diffuse swelling remained. Two months ago he first came to me for an otalgia on the same side. There was a little more lymphoid tissue just behind the Eustachian tube orifice on that side than I thought there ought to be. I probed it and it bled easily. A biopsy from this area was reported as lympho-blastoma. We clinched the diagnosis by eliminating the leukemias and Hodgkin's disease through a blood examination. Of course the neck swelling is now explained as an extension from the naso-pharyngeal tumor.

There is sometimes a hazard in relying on the laboratory findings. Last month I thought I was operating on an old chronic suppurative ear for a beginning meningitis. Successively there developed within the next week frank evidence of lateral sinus thrombosis, a cavernous sinus on that side and then on the other. The three operative efforts helped but temporarily. The significant point I wish to illustrate is that we would not get a positive blood culture until our third attempt, which was after the cavernous sinus had become involved. Whether the outcome would have been different, none can say, but I delayed tying the jugular 24 hours because of his apparently fine condition coupled with the negative blood culture.

I was interested in the speaker's conclusions about bleeding and coagulation time. I do not now rely on this test as a routine, but on the clinical history. Each adenotonsillectomy case is asked about any tendency to prolonged bleeding. Where there is any suspicion, the venous blood is tested rather than that from a superficial puncture. My chief trouble with tonsil bleeding seems to come from cases with undetected mild acute colds.

Lastly, a word about biopsy. I had recently read afresh that someone feared metastatic extension from taking a specimen. This patient had been under my observation for several months. The laryngeal tumor at the junction of the middle and anterior thirds of the true cord was growing slowly into the deeper tissues. He was about fifty, strong, healthy,

never any cough, showing a negative Wassermann, a negative sputum, a normal temperature, a negative chest examination, clinically and by x-ray, so I did a laryngo-fissure and carefully removed the affected cord with the tumor in the middle of it—without an initial biopsy. The pathological report came back—tuberculosis. In spite of my entreaties, the pathologist refused to find any malignancy.

Our English friends across the water have a convincing and sturdy way of doing things. We would profit if we could hear them more often. Two years ago Sir St. Clair Thomson honored the American Laryngological Association, as Mr. Herbert Tilley honors us today. His comments on the taking of a specimen for biopsy were interesting. He did not think that such a procedure in laryngeal carcinoma would cause an extension, but he reminded us of the difficulty of getting a good specimen, and that a negative report did not offer proof. Fifty per cent of his operations for cancer of the larynx had been done without biopsy. In the last analysis he felt that one's surgical judgment should decide.

Such are my reactions to Dr. Connor's careful paper, a copy of which I had the pleasure of reading day before yesterday. I feel sure that many will return home with added determination to go into these laboratory details. Not only will our work be more interesting, but our patients will profit. We are indebted to the speaker.

Dr. Ralph A. Fenton, Portland, Oregon: We are indebted to Dr. Connor for an authoritative monograph for ready reference, something with which we can awaken our own local pathologists to some of the newer things that we want done. A good many of them are in the habit of doing things in their own way and do not take kindly to suggestions from us laryngologists.

Because of the enormous size of the subject Dr. Connor did not of course attempt to cover it all; I would like to add, in regard to lumbar puncture, the fact that suboccipital cistern puncture has been found to help when lumbar puncture is inconclusive in very severe intracranial complications. It has been used a good deal by Lemaitre in France.

In respect to the examination of molds affecting the external meatus, I might suggest that in addition to the ordinary molds we should look for the ringworm fungus, which not infrequently is inoculated into the area from infestations of the feet, or of the genital region. We have found the use of a platinum loop for cultures a safer thing and one from which one can get more accurate conclusions than from the use of sterile cotton swabs. We have also found it very useful to employ Dean's double cannula in testing out the bacteriology of the maxillary antrum, rather than to rely upon the chance contaminations which are found by the ordinary puncture. Another thing we find useful in many of these bacteriological determinations is the use of blood agar containing human blood protein rather than blood protein from various animals. This is of course of value in the preparation of vaccines.

Another thing in which we should be after our pathologist friends is with respect to the various types of allergic determinations, as to the validity of the various series of skin tests, whether scratch or intradermal tests. I do not think we should rely upon the instructions sent us with

the ordinary list of allergic test proteins by the various commercial manufacturers.

I want to compliment Dr. Connor on his splendid monograph.

DR. S. J. KOPETZKY, New York, N. Y.: This encyclopedic paper must be read and re-read to be properly appreciated. I have very little to add except the suggestion that so far as we are possibly able we should do this laboratory work ourselves.

In the taking of blood cultures I would add that we should be careful not to impinge on the time when primary bacteremia takes place. It takes place in many conditions and has no very important pathological significance. It disappears in from ten to eighteen hours. It may interest you to know that you can get a positive culture if you take the blood right after a tonsillectomy. There is no pathological significance in this finding. The time at which you take the blood culture is of importance in estimating the significance of the findings.

In regard to cerebrospinal fluid, I want to stress the necessity of doing puncture under control of a manometer, reading your results, and watching the blood pressure of the patient while you are doing the puncture. This increases the safety of the procedure. Very often you should compare the result of a cerebrospinal puncture with that of a cisterno puncture, which it is now easy to do without anaesthesia. The chemistry of the cerebrospinal fluid should be read in relation to the cytology. Another point is that a study of the chlorine in relation to the lessened alkalinity of the spinal fluid gives you data of differential diagnostic value in the commencement of meningeal infection.

The improvement that has taken place in the use of laboratory aids in otology is great, but it is not sufficient. These aids should be used daily, so that comparison may be made and progress or retrogression noted. The examinations should be done at regular intervals and recorded. Only by a study of these fluid tissues of the body, as they change from time to time in the course of the disease, can a comprehension be acquired of the course the disease is taking, and both prognosis and differential diagnosis be aided.

Dr. Connor has presented one of the finest papers on this topic that it has been my pleasure to read.

DR. CHARLES E. CONNOR, closing: I have very little to add to what has been said. I would like to second what Dr. Spencer said about the inadvisability of waiting for positive blood cultures in cases of suspected thrombosis. There are so many other things that can be done in the way of study that will help us that I think it is an injustice to the patient to delay operation waiting for a culture which may never become positive.

As to the value of the clotting or bleeding time in tonsillectomy, I believe a careful history of the patient and a careful physical examination are of vastly more importance.

Dr. Berry spoke of the different interpretations which may be placed on biopsy specimens. It is a moot question whether or not biopsy should be done in suspected malignancy. Evidently the answer depends upon one's confidence in his ability to make a diagnosis without the microscope.

I agree with Dr. Kopetzky about the use of serial studies, that one should not be satisfied with one white count, one blood culture, one urinalysis, but should get a series of examinations so that the biologic reactions of one's patient may be known.

When one begins to go into the field of laboratory aids in the diagnosis of ear, nose and throat conditions one is struck by the number of points of contact between otolaryngology and general medicine, and the further one goes into such a study the more apparent the relationship becomes, the plainer it is that the man who is doing ear, nose and throat work is not capable of following out the various diagnostic procedures that he should use in his cases. He will have to know when his patient needs the advice and care of an internist; he will have to know particularly what diagnostic aids are of peculiar value in his own field. There are some aids which the ear, nose and throat man should know and be capable of carrying out; there are others which he cannot be expected to know, those that fall more particularly into the field of the internist. The point I would like especially to make is that the ear, nose and throat man should be always alive to the situation and call for diagnostic study by the internist when necessary.

I want to express my thanks to the discussers of this paper.

CONSIDERATIONS UNDERLYING TREATMENT OF SINUS DISEASE

EUGENE R. LEWIS, M. D.

LOS ANGELES, CALIF.

Among other branches of the practice of medicine, rhinology is very largely concerned with epithelial tissues. Rhinologic literature is filled with articles describing catarrhal, infiltrative, infectious, hyperplastic, degenerative, polypoid and mixed pathologies of the commoner inflammatory diseases of the upper respiratory tract; the x-ray and the bacteriologic reports appear prominently; surgical procedures are always accorded leading interest; diagnosis and differential diagnosis are occasionally given special attention; prognosis is generally discreetly relegated to the background or omitted entirely; results reported usually concern *operative* recoveries and shed little or no light upon the ultimate effects of treatment on the patient. What I have to say does not concern new growths, nor do I propose to ask anyone to follow me through the usual fields of discussion on the off chance of gleaning sufficient to warrant the trouble. This paper confines itself to experiences with "sinus troubles," and to certain deductions relating to epithelial tissues.

Radical surgery in "sinus trouble" has failed to justify itself; it does not rest upon a rational basis; it has been largely conceived in anatomic rather than physiologic ideations, and is fundamentally unsound. The past decades have witnessed a succession of operative procedures which have sprung from intensive studies of operative technique. The anatomy of approach and accessibility, and convenience to the operator, rather than the ultimate effect on the patient have been paramount considerations. There are probably few in this, or any other gathering of rhinologists who do not share to some degree in impressions of this general nature. My own post-operative contacts with patients almost uniformly confirm the disappointing results of sinus surgery; as a rule the original operation is early lost

sight of in its numerous progeny of unanticipated secondary operations and unrealized expectations—always about one lap behind the will-o-the-wisp cure,—which ultimately leads the patient, more or less exhausted physically, morally and financially into the arms of medical agnosticism or cultism. The lure of surgery rather than the welfare of the patient seems to be the only tenable explanation of the multiplicity of recent operations on the sinuses in the face of countless discouraging results. Once under way, the momentum of the process of these surgical evolutions was tremendous; therapeutic failures, numerous as they were, were scattered so widely as to exert but little deterrent effect. As a consequence, over thirty years have passed before signs of an arrest could be sensed. However, the end of the third decade of the twentieth century is witnessing a very definite revulsion against destructive surgery of the nose, a revulsion originating not among rhinologists but among patients and their medical advisors. Physiology rather than anatomy, is finally beginning to sway the therapeutic thinking of the rhinologist.

The common goal of all medical efforts, the well-being of the body as a whole, is the product of all the mechanismal factors entering into the life of the individual. Of these physiologic mechanisms—the respiratory, digestive, nervous, circulatory, and glandular systems may be looked upon as the greater factors, the muscular, skeletal, and fascial systems as the lesser factors, bearing upon general physical and mental well-being. Mutual interdependence and co-operation are the essence of working relations between them.

Physiology deals with material tissues under ordinary living conditions. The terms "well" and "ill" are almost as dangerous to use the terms "right" and "wrong," or "good" and "evil." Living involves endless contacts with environmental influences and adversities, and these interactions entail endless adaptations; successful adaptations, analyzed, turn out to have been mainly minor ills—successfully negotiated. The hand wields the spade and is blistered—an ill; with persistence it becomes calloused—"adapted" to use the spade. The sun beats upon the skin, blistering it—an ill; with persistent exposure it becomes tanned—"adapted" to withstand the sun's rays. The sea-level dweller falls ill in the thin air of Pike's peak; continued exposure brings about red cell concentration permitting him to exercise as usual with no ill effects—"adaptation" to altitude. Bacteria in-

fect tissue—an ill; with continued contacts resistance rises conferring immunity—"adaptation" to infection.

We are compelled to maintain an open mind concerning the results of contacts between body cells and pathogenic organisms. Pathogenesis is not as simple as conjunction of cells and bacteria, in spite of the fact that it frequently seems so. The first wave of influenza which struck the Philippines in 1918 caused a widespread epidemic with no deaths. Six months later another influenza wave followed with very high mortality. The high mortality was determined to be due to the admixture of streptococcus, which had been absent in the previous wave. But those who had succumbed to the first epidemic were not affected by the second. Evidently the immunity gained by their recovery from the mild, unmixed influenza infection served to protect them from the second more virulent epidemic. The results upon the spirochaetal infection that have followed the incidence of malaria, the effects of tryparsamide, the clearing up of chronic tympano-mastoiditis on the heels of recovery from severe erysipelatous infections, the activations of latent tuberculosis and encephalitis by small pox vaccinations, the complete cessation of bronchial asthma during and after virulent febrile infections and many similar frequently noted departures from the usual in infectious pathogenesis are too pointed to be ignored. It seems fairly established that upon opportunities presented during allergoid tissue-reactions bacteria frequently find foothold in an individual otherwise not susceptible to successful attack. While it is difficult to see clearly the inter-relations between cellular reactions and their unusual effects on pathological processes it is certainly far from true that infection is always an unmixed evil or that avoidance of infection is always a physical boon. These are but a few examples of unexpected eventuations in adaptation processes.

Under modern conditions of living one must recognize in early childhood a period of several years, approximately between two and nine, as the time of average (or normal) child's beginning world contacts with foods, with environments, with indigenous infections. It is by no means true that every child manifests reactions to unprecedented environmental contacts either in the same way or at the same age; among these adaptations, immunity acquisitions include the so-called "infectious diseases of childhood"—but a list of the exanthemata by no means completes the infectious catalog. It is safe to say, however, that a large part of

the first decade is included in this period of immunizing contacts, and practically all normal children undergo with more or less impunity these gradual and natural immunizations. Upon his acceptance of some such fundamental conception of a period of what might be qualified as "normal illnesses" must depend the attitude of the physician in shaping up diagnoses and deciding upon treatments. Many children undergoing natural contacts with tuberculosis, streptococcus, stahylococcus, pneumococcus and other wide-spread strains of infectious organisms manifest slight and indefinite groups of symptoms, falling into the "febricula" category; others manifest symptoms referable to respiratory, digestive, or both, tracts; many fall into the category of "adenoid and tonsil troubles," with or without lymph-node affections. During these constructive episodes of more or less critical nature, traumata are sometimes poorly borne and affections of apparently unrelated and remote tissues are encountered—such as pyelitis, tenosynovitis, myositis, chorea, endocarditis, aprosexia, eczematoid skin and scalp lesions, and various allergoid reactions. Normally, these immunizing reactions of childhood are destined to successful termination by the earlier years of the second decade. Watchful conservatism and general supportive and protective care, with scrupulous attention to exceptional conditions demanding special measures of treatment, is the outstanding medical necessity during these years. The practice of seizing upon the upper respiratory phases of immunity acquisitions as opportunities for radical surgical measures cannot be too strongly condemned. Surgical removal of those nasal structures which at times give local expression to general immunizing reactions or to special sensitivities is in no way indicated or desirable, either from the standpoint of logical applied pathology or of therapeutic experience.

Before the age of maturity the individual has demonstrated with what success his own adaptations have proven able to cope with the difficulties encountered, and his "norm" can be fairly determined. These fundamentals of human natural history, considered in conjunction with individual history, and diagnostic study, will crystallize decisions as to what to attempt when confronting a problem; and therapeutic measures fitting into the picture will naturally suggest themselves. Except in rare instances, the value of bacteriologic and x-ray findings in the commoner upper respiratory inflammations is confined to confirming

details of the physical diagnosis, and they should not be exalted to the role of indicating the surgery. For example, nothing could be farther from the point than to construe extensive pneumatization revealed by x-ray as the determinant finding indicating surgical attack. Successful local reaction is usually directly—not inversely—proportionate to extent of pneumatization, other resistance factors being equal.

In general, surgical measures are based upon attempts at *drainage* or to *remove infection*. "Drainage," perhaps the most valuable of all surgical procedures, implies giving exit to something fluid rather than solid; and it often infers dependence upon gravity, as drainage openings are usually made in the most dependent portion, with provision for continuous drainage for a longer or shorter post-operative period. Normal drainage of the sinuses does not depend on gravity; the movement of secretion occurs entirely by reason of ciliary action—equally well against gravity as with it. Acute inflammatory conditions may impair ciliary action to such an extent as to stop emergence of secretion from a sinus. It is far from certain that this is disadvantageous, and it is certain to be only temporary. Gravity alone is unable to overcome the effects of viscosity and atmospheric pressure in effecting the exit of even gross quantities of abnormal secretion from sinuses. Equalization of air pressure within the sinus and the naris is necessary if fluid is to emerge from a sinus, whether moved by gravitation or ciliary action. Surgical ablation of infected tissues offers no prospect of removing infection from the upper respiratory tract; no matter how wide of the manifest infection limits such removal is made, the entire field is certain to be re-infected within a few hours, and surgery is bound to open new portals, perhaps dangerous ones, to infection.

For years a world wide search has been on for a "Therapia Sterilisans Magna." Various chemical, physical, bacterio-phage, electrical, protein, febrile, or other measures to attain this long-sought boon have proven partially successful. As in the beginning, even now, the most efficient single factor against infection is found in the natural reactions of the tissues themselves. Whatever of encouragement has been encountered in the application of other anti-infectious measures has not been under conditions excluding natural tissue reactions, and must still be evaluated as in conjunction with them.

All details (even those of seeming unimportance) of physiologic mechanisms are by no means chance formations; each stands for efficiency, each embodies thousands of years of natural selection and development. Interdependence between body mechanisms is certainly of extent and importance beyond our ken. By the working test of actual performance, man's physiologic equipment has proved its worth in making these successful adaptations without which would have been impossible his continued existence in the face of all the adversities since time began. None of the mechanisms which have contributed to this result may be looked upon as negligible; certainly man should regard them with respectful humility, seeking to understand, rather than deprecating, what he fails to evaluate highly. It is safe, despite the limitations of our understanding, to consider each mechanism as directly or indirectly essential to the body as a whole; and to consider natural cellular and structural details as the most efficient.

Bearing in mind the foregoing generalities, let us consider the epithelial structures of the upper respiratory tract. It is assumed that, as physicians, our goal is the optimum health and strength of man, that health and strength are the outcome of satisfactory adjustments to environments, that adjustments to environments are subserved by physiologic mechanisms and that functionations of a physiologic mechanism depend upon highly specialized cells so arranged as to be capable of efficient co-operation as a physiologic apparatus.

Studies of cell life establish that cells affected by, or injured by toxins or trauma possess considerable measure of recoverability with preservation of function, through reactive processes involving not only self-contained potentialities of the affected cells themselves but also inter-dependent reactions of other remote cell groups concerned with resistance and repair. Such highly specialized cells as fail to recover are thrown off, their places being taken by cells of lower order and inferior functionabilities. In a physiologic mechanism substitution of low order cells for highly specialized cells degrades the functional efficiency of the whole mechanism in direct proportion to the extent of such substitution.

Infectious, allergenic and other inflammatory and pathologic processes are but details of body reactions to environmental adversities. All contacts with environment take place through epithelial cells; the outcome of such encounters depends in great measure upon the individual cellular equipment and its mechani-

cal arrangement, whether advantageously or disadvantageously disposed not only as to local resentment and reaction to insult, but also with respect to facility of utilizing aid from the more remote mechanisms of resistance in terminating each episode promptly and successfully. Daily life comprises an endless series of encounters between epithelial tissues and enemies; unbroken health means that in these encounters the tissues succeed in maintaining the upper hand; illness means failure to maintain the upper hand. With the onset of illness not only the epithelial and sub-epithelial structures, but every body tissue capable of contributing to resistance concentrates upon the restoration of supremacy to the attacked tissues. Calls are issued mobilizing the defensive resources of the body; agglutinins, lysins, opsonins, anti-toxins, leukocytes,—all pour forth in prodigal proportions; endocrine, glandular and organic activities are speeded up, the general metabolic rate rises, with fever, tachycardia, anorexia, acidosis and dehydration giving evidence of depletion of the stores of glycogen, phosphorus, calcium, water and fat; nutritional and digestive activities and other processes of less than immediate usefulness in defense are slowed down in diverting all possible energies to the emergency job. Occasionally defensive activities of the tissues are further impaired by accumulations of necrotic detritus or toxic by-products in such amounts or locations as to embarrass by their presence already hard-pressed body cells; when the character or amount of these accumulations happens to be particularly vicious they may attain such proportions as to tilt the scale toward fatal outcome. Under these conditions, timely and wise surgery may afford critical relief and turn the tide of battle; but the surgeon must bear in mind the impetuous life-saving crew, who in their eagerness to fire the precious life-line to the vessel on the rocks, trained the mortar too hastily, and when the smoke had cleared away, found they had shot the last man off the mast.

Epithelial tissues are of prime importance in encounters between the body and disease-producers; they are the "shock-troops." Destruction of highly specialized epithelial cells represents permanent impairment of essential equipment. Not only the cells themselves, but their advantageous disposal in relation to equally important sub-epithelial lymph lacunæ, integral and regional lymph nodes, efferent lymph ways, general circulation contacts with remote cell-groups and conversely all their afferent

facilities for effecting co-operation on the part of systemic resistances, must be regarded as demanding our utmost efforts towards protection and conservation.

Treatment must be based upon considerations of the future welfare of the patient as well as of the present emergency, in dealing with inflammatory diseases of the upper respiratory tract. It should be unthinkable to purpose surgical destruction of essential tissues because these tissues appear at the moment to be threatened by some pathologic process. The lateral nasal wall tissues are the essential portions of a vital organ of no less importance than the kidney or the liver. The determination of cellular viability under infectious attack should be left to the cells themselves. Surgery may well limit itself to measures calculated to relieve intolerable local situations with the utmost conservation of essential cells and structures.

DISCUSSION.

Dr. Arthur W. Proetz, St. Louis: Dr. Lewis and I are again off on the subject of conservatism versus radicalism.

I wish to emphasize that the most important thing to be considered is the patient as an individual and not as an antrum having epithelial cells which will or will not repulse infection. After all, we are treating the patient to give him the greatest relief over the longest period of time. It is not a matter of next month, but a matter of the rest of his life.

Dr. Jonathan Wright used to say that he had never seen a case of acute tonsillitis artificially produced by inoculation of an unbroken mucous membrane with any sort of bacteria. Therefore the mere presence of bacteria is not all important. The thing we want is a healthy patient and not one without bacteria. It seems to me it is useless to free a nose from bacteria and have a dry, clean mucosa but an uncomfortable patient. It is much more desirable to have a fairly good nose in a healthy person, than a nose which from a cosmetic standpoint is a clean nose, and a patient that is extremely uncomfortable.

A CONSIDERATION OF DISEASES OF THE BLOOD AND LYMPHATIC GLANDS IN RELATION TO OTOLARYNGOLOGY

PERRY G. GOLDSMITH, C.B.E., M.D., C.M., AND GREGOR MCGREGOR, M.B.

TORONTO, CANADA

It is written that there abideth Faith, Hope, and Charity, but the greatest of these is Charity, so Robert Hutchinson, an eminent British physician, in aptly applying this old but familiar text to medicine, makes diagnosis a of matter of faith, prognosis a matter of hope, and treatment a matter of charity (too often so), but the greatest of these is diagnosis.

It is only by the intelligent interpretation of signs and symptoms that diagnosis reaches a firm foundation. It is all important if the treatment is to be intelligent and efficient. In fact, the first part of the treatment is diagnosis, the second is diagnosis, and the third is diagnosis.

In this interpretation of signs and symptoms the laryngologist has frequently to deal with bleeding from the nose and throat, and altered states of the glandular system shown in enlargements of the glands, such as those of the Waldeyer Ring, and the cervical neck chain. As such pathological conditions rest primarily on alterations of the blood, or blood-forming organs, we considered a study of case records showing the important and close relationship otolaryngology has to general medicine would be of interest and instruction. It must be remembered that the correct diagnosis of many obscure cases of haemorrhage from the mucosa of the nose and throat can only be made after skilled analysis of the completed blood picture. Often only the microscopical examination of a section of the gland affected can determine the underlying disease, but even skilled pathologists frequently have great difficulty in arriving at a diagnosis—for example, in cases of infective mononucleosis with ulceration of the tonsil.

In looking through the case records of the Toronto General Hospital, in those dealing with altered blood states in which oto-laryngology has a part, it seemed simplest to divide them into several main groups: one in which bleeding was a prominent factor, another in which glandular enlargement seemed to dominate the picture, and still another in which neither of these signs was prominent and which we have taken the liberty to call the anaemic group.

Group 1. (a) Congenital telangiectasis.
(b) Polycythemia vera.
(c) Myeloid leukemia (chronic and acute).
(d) Lymphatic leukemia (chronic and acute).
(e) Symptomatic purpura.
(f) Purpura haemorrhagica.
(g) Hemophilia.
(h) Salvarsan and benzol-poisoning.

(a). In congenital telangiectasis there is a history of repeated and severe epistaxis for many years or even from childhood. Gradually weakness becomes prominent and only then does the patient seek medical advice. By this time anaemia is very marked, the hemoglobin being as low as 20%. The clotting and bleeding times are normal so any necessary operation may be undertaken with safety. The diagnosis rests on the finding of reddish-spider-like telangiectic spots in the mucuous membranes and skin which come in crops and fade on pressure. These spots are most commonly found on the septum in the region of Little's area and on the anterior portion of the turbinates. They are frequently on the tip of the tongue, and may be seen in the naso-pharynx, larynx, and even on the tympanic membrane. Those on the septum bleed quite easily and careful cautery or radium therapy does a great deal for the comfort of the patient and gives the blood-forming organs a chance to recover.

(b). The florid cyanotic individual who presents himself with a history of repeated epistaxis should have a blood examination, for if he has a red blood count considerably above normal and an enlarged spleen, he is likely to be a case of polycythemia vera, provided there is no physiological cause for the increase of red blood cells. The blood may even be so thick that it is difficult to get sufficient plasma for purposes of examination. Consequently death by thrombosis is not an uncommon termination.

(c). Not uncommonly in acute myeloid leukemia there is bleeding from the nose or mouth with ulcerations. The patient is very ill, with considerable elevation of temperature, and marked splenic enlargement. The glands of the neck are usually enlarged. The white blood count may not be very high but will show a large percentage of very immature forms such as myeloblasts, and a marked secondary anaemia is present.

In the more chronic forms the spleen is also very large, but there is a very marked increase in the white blood count with a smaller percentage of immature forms. Bleeding from the mucous membranes comes in the terminal stage.

(d). Acute lymphatic leukemia resembles an acute infection with fever, swelling of the tonsils, ulcertative angina, and cutaneous haemorrhages. The glands of the neck enlarge and usually those of other groups, but the spleen is not very large. The blood picture with its high white blood count and large percentage of large lymphocytes makes the diagnosis. The acute types of leukemia are very similar and it may be quite difficult to differentiate. However differentiation is not important for the progress in either case is rapidly fatal.

In the chronic forms of this disease small lymphocytes predominate the blood picture, there is a general glandular enlargement, and the patient is not very ill until the later stages.

(e). In symptomatic purpura it must be remembered that the ecchymoses and haemorrhages from the mucous memberane are only symptoms of some underlying condition, infectious, toxic, cachectic, severe anaemic, neurotic, or mechanical. The actual cause can only be determined by a careful and complete examination of the patient, and treatment must be instituted accordingly.

(f). Purpura haemorrhagica is a more severe form and may be rapidly fatal, with death from loss of blood, or from haemorrhage into the brain. It differs from the other purpuras in that the blood platelets are diminished in number. The bleeding time is much prolonged, the blood clot is soft and non-retractile, while in haemophilia bleeding time is not prolonged, the blood platelets are normal, and the clot retracts normally. The coagulation time in haemophilia is markedly prolonged, while in purpura it is normal or nearly so. The application of a tourniquet to the upper arm results in the formation of petechiae on the forearm

in purpura but not in haemophilia. Bleeding is most common from the nose and vagina.

(g). In hemophilia it is essential for a diagnosis that the individual should have been subject, more or less, to bleeding from various parts of the body throughout life. Haemophilia without demonstrable inheritance is rare, is not found in females, and is transmitted only by them.

(h). Agranulocytic angina. A toxic patient with sore throat, fever, chills, bleeding, sloughing ulcerations in the mouth and with a fetid odor to the breath may be mistaken for septicaemia, but if examination of the blood shows the polymorphonuclear neutrophiles to be almost completely wiped out, the case is likely to be agranulocytic angina. The lesions usually begin as small superficial ulcerations covered with a greyish or yellowish exudate which on removal leaves a bleeding surface. These may enlarge and increase in number very rapidly until they become large burrowing ulcers. The most common location is on the tonsils which are usually inflamed and enlarged and they may become so large as to make swallowing difficult. The pillars, uvula, soft palate, tongue, lips and even the gums may be involved. Even where the symptoms are not so marked one must be careful in giving a prognosis for the outlook is almost hopeless.

One must be careful, however, that he is not dealing with another condition associated with an agranulocytic symptom complex such as severe sepsis, aleukemia, leukemia, pernicious anaemia, poisoning with benzol or salvarsan, and the effects of X-ray or radium therapy.

(i). Salvarsan poisoning may produce bleeding, sloughing ulcerations in the mouth associated with a diminution of the polymorphonuclear neutrophiles, but the history of treatment prepares the way to a diagnosis.

Group 2. (a) Infections.
(b) Malignancy.
(c) Lymphatic leukemia.
(d) Aleukemic leukemia.
(e) Lymphosarcoma.
(f) Hodgkin's Disease.
(g) Infectious mononucleosis.
(h) Tuberculosis.
(i) Vincent's angina.
(j) Actinomycosis.

(k) Carotid body.
(l) Fibroma.
(m) Lipoma.
(n) Tonsils.

(a). Local enlargement results most frequently from focal infections by way of the lymphatics so that the area drained by the particular group enlarged must therefore be searched for the portal of entry. In an adult in the absence of an obvious inflammatory focus the possibility of metastatic deposits must be considered.

(b). Seeing that malignancy of the mouth and oesophagus is a frequent disease, the so-called sentinel gland in the neck in carcinoma of the stomach must be kept in mind. Glands in the lower cervical group on one side of the neck are frequently the first to be enlarged in Hodgkin's Disease.

In generalized enlargement where two or more groups are involved the causative agent may be conveyed by the blood stream, as for example, in infectious mononucleosis rubella, septicaemia, secondary syphilis, or bubonic plague. In this way the generalized enlargement has a definite diagnostic value.

If the glandular enlargement has arisen insidiously and if the patient's condition, generally, is not acute, the probable explanation lies in the direction of such disease as leukemia, Hodgkin's Disease, and rarely, tuberculosis and sarcomatosis.

The specific features of glandular enlargement calling for special note are: the number and distribution within the group, the size, mobility and consistency, sensitivity, whether the glands are discrete, or coalescing, and involvement of the skin, or surrounding structures. When the glands are adherent to one another or to surrounding structures, it means the disease process has spread through the capsule, as it does in tuberculosis, pyogenic infection, and malignancy. In chronic cases with several groups involved, the disease is likely to be chronic leukemia, Hodgkin's Disease, or rarely lymphosarcoma. Leukemia gives us the least difficulty in diagnosis because of a distinctive blood picture.

(c). The chronic lymphatic type of leukemia is characterized by generalized glandular enlargement, and a blood picture showing a high white cell count 100,000 or more, in which the great majority are lymphocytes. The spleen may also be enlarged, although a large spleen and small glands is more characteristic of chronic myeloid leukemia, where the majority of the cells are

of the myelocytic type. The individual nodes in a group are of much the same size, mobile, discrete, soft, and show no tendency to break down or involve the skin or other structures. They are insensitive to palpation and cause no discomfort apart from their size.

(d). The aleukemic type shows a large glandular enlargement without a high white count, but with a relatively large percentage of lymphocytes, as in lymphatic leukemia. Tonsils are often enlarged both in the lymphatic and aleukemic types.

(e). In lymphosarcoma the tendency is for the glandular enlargements to be unequally transformed into large masses, the individual nodes showing marked variation in size. Their mobility is limited and there is a tendency for the whole mass to be adherent to the deeper structures. Progressive enlargement of both tonsils is more likely to be lymphosarcoma than Hodgkin's disease. Pruritus, fever, and sweating are absent, metastases are uncommon, and the spleen is not enlarged. Biopsy may be an aid in diagnosis.

(f). Hodgkin's Disease. This often begins with enlargement of the glands in one side of the neck but it may begin in any group. The first symptom may be obstruction of the bowel due to enlargement of the lymphoid tissue in that location, or cough, or dyspnoea from enlargement of the mediastinal glands. Swelling of the arm or abdomen may be due to pressure on the venous flow, or enlargement in the groin may simulate hernia. There is a tendency towards an orderly spread from one group to another. The glands are discrete, rather firm and elastic, not tender and do not tend to involve the skin, while in metastic carcinoma they are hard, irregular and fixed. Progress may be slow or rapid. The blood picture is not distinctive though there is often an increase in the endothelial leucocytes. There is a great tendency towards infiltration of other organs. Fever, pruritus, and sweating are common, and the spleen is usually enlarged.

Biopsy may be an aid to diagnosis, but the interpretation of the microscopical picture is often difficult. The typical picture is an increase in fibrous tissue with a disturbance in the topography of the gland and the presence of multinucleated giant cells and eosinophiles. In these cases removal of tonsils is not going to cure glands in the neck.

(g). Infectious mononucleosis is a self-limited disease of young adults, usually males, characterized by an acute onset with fever,

malaise, sore throat, and glandular enlargement which usually begins in the neck. The tonsils are often inflamed and may be ulcerated. There is some increase in the white blood count but the greatest change is in the percentage of lymphocytes which is very high. This confuses the condition with lymphatic leukemia, but the acute onset with fever, sore throat, and relatively low white blood cell count makes the diagnosis. In a few weeks the patient is back to normal and all anxiety is relieved. Biopsy is likely to be very misleading as the picture is one of lymphosarcoma. As regards biopsy Professor Duncan Graham, Professor of Medicine in the University of Toronto, makes the following statement:

"Among clinicians the impression is too widespread that the excision of a gland for microscopical examination is essential in the diagnosis of superficial glandular enlargements. This method for making a positive diagnosis has definite limitations. Too often the clinician advises the removal of a gland for microscopical examination without realizing that it is impossible for the pathologist to differentiate by histological examination between glands from cases of lymphoid leukemia, aleukemia, lymphosarcoma, or certain cases of Hodgkin's Disease, without a report of the clinical examination of the patient, and the results of the hematological examination of the blood. This information alone is usually sufficient for the making of an accurate diagnosis. The microscopical examination of an excised gland is chiefly of value in the diagnosis of atypical cases of tuberculous adenitis."

(h). Tuberculous adenitis is most common in children and is associated with disease of the mucous membranes or enlargement of the tonsils. It is most common in districts where the milk is not pasteurized. The glands usually begin in the submaxillary group, and go on to form large knotted masses to which the skin becomes adherent. Inflammation and suppuration may occur. Pruritus is absent and the spleen rarely becomes as large as it does in Hodgkin's disease. It is exceptional to find general tuberculous adenitis. If diagnosis is in doubt a gland may be removed for examination.

(i). Vincent's angina may produce marked enlargement of the cervical glands with tenderness, but the presence of deep ulceration, usually in the tonsil, from which the causative organisms can be found will make the diagnosis. However, the ulceration

due to malignancy must be kept in mind for the Vincent's organism here may be a secondary invader. If there is any doubt it can be settled by biopsy.

(j). Actinomycosis may involve buccal, lingual, or pharyngeal structures. It produces ulceration and marked infiltration with secondary enlargement of the cervical glands. Diagnosis is made bacteriologically, the causative organism being a streptothrix.

(k). At the level of the top of the thyroid cartilage occasionally there is enlargement of the gland known as the carotid body. These tumors are at first benign but may become malignant. The situation and the fact that it is a single enlargement give a clue, but diagnosis is only made by biopsy. It may be confused with (l) fibromata and (m) lipomata.

(n). A patient with a story of progressive enlargement of one tonsil or both is entitled to an examination which will exclude malignancy, though it may be quite innocent and due only to a chronic infective process.

Group 3. Anaemic group.

(a) Pernicious anaemia.
(b) Secondary anaemia.
(c) Chlorosis.
(d) Hemolytic jaundice.
(e) Banti's disease.

(a). A patient presented himself with the story that for several months previous he had been bleeding a great deal from the nose and that his eyesight was becoming much impaired. Careful examination established a diagnosis of pernicious anaemia and his failing eyesight was due to old and new retinal haemorrhages. His left vocal cord was almost stationary and there was a paresis of the internal tensor muscle. Careful examination by the medical department established a diagnosis of pernicious anaemia and proper treatment restored his voice and larynx to normal.

Other cases have various complaints such as sore throat, sore tongue and headache. A blood and gastric analysis will establish a diagnosis and save the disappointment of local treatment to nose and throat.

(b). Removal of tonsils is not likely to bring back the loss of bodily and mental vigor to the individual suffering from a marked secondary anaemia, the cause of which may be in the rectum.

(c). Neither is it likely to cure the green sickness or chlorosis of young blondes who come from the factories of Europe.

(d). In Banti's disease the treatment of haemorrhages from the mucous membrane may be the removal of the spleen, for this condition is marked by bleeding from the mucous membranes, enlargement of the spleen, and a secondary anaemia associated with a leucopenia.

(e). In long-standing cases of hemolytic jaundice haemorrhage may be a prominent feaure, but the primary factor in the disease is an increased fragility of the red blood corpuscles.

Not uncommonly cases with a haemorrhagic diathesis complain of deafness or vertigo. There is no reason why there can not be a haemorrhage into the labyrinth as well as elsewhere and this may be the cause of symptoms. This can only be ascertained by labyrinthine tests and blood examination.

A few typical cases to illustrate may be of interest:

1. A middle-aged woman, the mother of a medical student, was admitted to the hospital with a history of having lost a great deal of blood following the extraction of teeth. It was scarcely possible to see much of her face or anything in the mouth because of blood, and the results of styptic medication. Careful examination revealed a very large spleen and a red blood count of over eight million, a case of polycythemia vera.

2. A woman aged 46, was cyanotic from obstruction to respiration, thought to be due to tuberculous inflammation of the larynx. Laryngeal examination revealed a normal larynx, but careful x-ray examination in the lateral position disclosed a very large mass in the mediastinum. This was believed to be Hodgkin's Disease, and x-ray therapy reduced the mass and the respiratory distress disappeared.

3. A young man aged 23, was sent to a sanatorium for investigation of the lungs because of fever, pallor, and loss of weight. On arrival he developed pain and swelling in one cheek and involvement of the maxillary sinus was considered. The otolaryngologist was suspicious of a blood dyscrasia. He was able to palpate a spleen markedly enlarged and blood examination revealed an acute myeloid leukemia.

4. Another man in a very important position was seeking relief from a progressive weakness. He had also had repeated epistaxis. He had had his blood examined, and was told it was normal or even better than normal. The otolaryngologist who was consulted for the epistaxis noted the cyanotic appearance and asked for a complete blood examination. It was a case of

polycythemia vera with a red blood count of ten million. Venesection, x-ray treatment to the long bones and the administration of phenylhydrazin reduced the count.

5. A man presented himself for examination complaining of pain in the ear and swelling in the neck. He had large diseased tonsils but at the base of the tongue there was a small ulcer which proved to be malignant and was the cause of his symptoms.

6. A medical student was ill with headache, fever, malaise, and sore throat. His tonsils were large and inflamed, and there was a sloughing area in the upper pole of the right. A biopsy was done and the report was lymphosarcoma. However, in two months he felt as well as ever. It was a case of infectious mononucleosis.

In this paper no effort has been made to cover all the differtial diagnostic points in these blood and lymph gland dyscrasies. It has been our endeavor to emphasize the importance of the medical examination necessary in otolaryngological patients in whom our cross-examination points to the possibility of a general systemic disease rather than a local disorder; but our consideration has not gone beyond the title of the paper. We have, therefore, made no reference to such conditions as hypertension, chronic hepatic, or renal disease, uncompensated heart disease, infectious fevers and local lesions in the nose.

We are indebted to the Medical Service of the Toronto General Hospital for their cooperation and for the use of their records.

DISCUSSION.

DR. JOSEPH C. BECK, Chicago, Ill.: The ability to discuss this paper, especially after such a wonderful feast in the presentation of papers dealing with a similar subject is not possible for me, seeing that I was not fortunate to see Dr. Goldsmith's paper before he read it. That is not his fault but my own.

To me, personally, this presentation as well as the other paper on laboratory aids, is a tremendous satisfaction after nearly twelve years of carrying on examinations, first for the Academy and then for the Board. As the men come up to be examined in the subject of pathology we immediately hear the statement, "I send my stuff to the laboratory," as if that were an excuse for not knowing the things that are presented here this afternoon. While we may not do our own laboratory work, and most of us do not, at the same time when the laboratory man sends us his report we should be able to evaluate the statements he makes.

The number of conditions which Dr. Goldsmith covered in his paper which are general diseases with local manifestations, is so large that to discuss any one of them would take too much time, so I will limit my remarks to a few points in the question of haemophilia. He brought out the point as to male and female having this condition. Let me state a case that will illustrate this point.

I had a patient that developed, following childbirth, a purpura haemorrhagica. This patient had had an infection of the throat for a number of years and I had cared for the throat condition. She had a very severe postpartum hemorrhage, recovered, but continued to have repeated nasal, mouth and gum hemorrhages. It happened that Prof. His of Europe was in this country and saw this patient in the hospital, in a very extreme condition, and advised the use of fresh blood, placental blood, dropped on the hemorrhagic spots in the mouth, and it was a great surprise the effect it had locally. That case led to a great deal of study on this subject.

Subsequently I had two girls who had acute otitis media, with records of bad tonsil and adenoid conditions, and the pediatrician insisted that the tonsils and adenoids be removed. Knowing what Dr. Goldsmith has told us as to how haemophilia exists, I operated these two girls, and in spite of the fact that we know how the female is exempt from that disease, there was no appreciable difference in the bleeding, although the clotting time was very much delayed. I therefore hesitated about operating and gave the patients all sorts of treatment, but there was no effect on the bleeding.

There is one general disease with local manifestations that I am sorry the doctor did not mention, and which we as laryngologists see first. I have seen three cases, and the doctor now has reported a good many more, of pemphigus of the pharynx and larynx with definite blood changes, such as are seen in agranulocytic angina.

Excision of the gland of the neck is perhaps not of specific value in diagnosis of many of the diseases, but when you remove a gland and show that it is tuberculous or malignant, I think it is of value. In the other blood diseases with glandular manifestations I agree it is not. I enjoyed this paper and all the others this afternoon.

Dr. Mark J. Gottleib, New York City: Dr. Beck voiced my sentiments when he said that the paper covers a great deal of general medicine which it is impossible for me, at least, to discuss.

The question is often put to the rhinologists—Why is it that bleeding occurs after throat operations, particularly tonsillectomy, and does not occur so frequently following operations in other parts of the body? We have tried to find an answer to that. One individual who seemed to be very well posted on the subject suggested that the glandular tissue in the throat in Waldeyer's ring has a definite relationship to other hematopoietic organs, and that if a sufficient compensatory reserve did not exist after removal of the tonsil, bleeding would occur. I am not talking about those cases where we have abnormal blood findings; I am talking about cases where the coagulation time is normal, the bleeding time is normal.

My presumption is that Dr. Goldsmith presented this paper with the idea that we should be aware of the conditions which he enumerated in coming to a conclusion as to the advisability of operative interferences in

certain types of cases and how to handle such cases when they present themselves.

As to delayed clotting time, some years ago Dr. Seymour Oppenheimer and myself did some work on prolonged bleeding time and had a lot of children examined before operating. In some family groups where the time was prolonged we injected whole blood from the mother and father and then the children developed normal bleeding time before operating. Very little bleeding was then encountered. In some cases with prolonged bleeding time we decided to operate without doing anything to the patient and obtained just as satisfactory results.

FOREIGN CLINICS

SECORD H. LARGE, M. D.

CLEVELAND, OHIO

After one has practiced his specialty for a certain number of years, there comes to him a longing to take stock of himself. It was for this reason, along with a desire to work out some original problems that the author spent a year in the Ear, Nose and Throat Clinics of Europe. There are many advantages in a year's sojourn. It gives you an opportunity to become better acquainted with the surgeons in the different clinics and you are able, not only to follow them in their clinical work, but you are invited to witness many interesting operations on their private patients. Such surgeons as our distinguished guest, Mr. Tilley, extended to us this courtesy.

The greatest advantage is that you are able to see the surgeon's end results. To get the most out of such a trip one must go with a receptive mind and not one of criticism.

We visited one clinic in Europe and complimented the operator on the skillful performance of a very difficult operation. His reply was: "Thank God for visitors that are not always telling you how they did it in their country."

We would like to sell you the idea of taking a year off and sojourning with our brethren across the waters where you would have one of the most profitable and happiest years of your life. Wherever we went, we were so graciously received.

We were surprised and delighted to find that in not only the larger clinics, but also in some of the out of the way clinics, how well informed they were in the work of our American confreres.

We wish to add here that we had the honor and pleasure of being present when our President, Dr. Mosher, was made a member of the British Laryngology Society.

It would be impossible in the time alloted, to give a complete survey of the interesting work seen in the different Ear,

Nose and Throat Clinics of Europe, so will, as briefly as possible, outline some of the most interesting subjects as they appeared to us.

One thing that impressed us was the attitude of the surgeon to the clinical patients. Everywhere they were treated with the utmost kindness, gentleness and consideration. The clinics appeared to us to have a larger attendance and the class of patients was very different from our previous visits. They appeared better dressed and better nourished than formerly. To a visitor, it seemed as though the clinics were being imposed upon judging from the appearance of the patients and from the large number arriving in automobiles, but the surgeon never seemed to question whether he or she was worthy of his time and skill.

Anesthesia

We saw the best anesthetists in the British Isles. They limit their practice to anesthesia and are all licensed practitioners. While they are trained in all forms of anesthesia, we thought their form of intra-tracheal anesthesia was the best we had seen anywhere.

Most of their anesthetics are started with nitrous oxide or more often, ethyl chloride, followed by ether and chloroform. At first chloroform appeared to us to be dangerous, but after seeing it administered in hundreds of cases without any fatality, we changed our views. It has many advantages in operations about the head as it is mixed with oxygen and ether and a very little seems necessary to keep the patient completely under. There is certainly less bleeding and less mucus with which to contend. They claim that they have fewer post pneumonias following it than when straight ether is used.

As you know, practically all their operations are performed under a general anesthetic. We saw only three surgeons in the British Isles practice local anesthesia. Ethyl chloride is used extensively for minor operations. In Edinburgh, practically all tonsillectomies and adenoidectomies in children are performed under it.

Note: We consider it a very safe and efficient anesthetic as we have now used it in over six thousand cases without a fatality.

Etheylene we never saw used. Atropin without opiates, is administered in practically all cases. In operations around the face, castor oil, as a routine, is instilled in the eyes. Carbon di-

oxide was administered immediately following the operation. They think it prevents atelectasis.

On the Continent, local anesthesia is used oftener than general. Novocaine with adrenalin, was the popular method. Some administered morphine alone or combined with scopolamin—the best results from the latter were when the patient was placed in a darkened room with absolute quiet. Some bandaged the eyes but the majority did not.

Asepsis

We found good asepsis in almost every clinic we visited. In most clinics, visitors had to don cap, gown and mask, and, in some cases, rubbers. Most of the surgeons used spirits as an antiseptic to sterilize the face. Some used 1-2000 biniodide mercury in spirits. Tincture of iodine or picric acid were used in most of the clinics to paint operating surface.

In plastic surgery, mastisol, a form of varnish, is used to stick towels to the skin and thus block off the field of operation. It is easily removed with benzine.

Tonsillectomy

We saw over one hundred surgeons perform tonsillectomies and it seems we saw about every different form of operation. In the dissection operation we saw many surgeons complete the operation without the use of the snare.

In the British Isles, general anesthesia is used in practically all cases. Lung abscess, following tonsillectomy, is practically unknown and we think the reason for it is due to the position of the patient, during and after, operation. In practically all the clinics visited, the patient was placed on his back with a large pillow under his shoulders, the head being in the occipito atloid position and with the surgeon seated at the head of the table.

Universally they use Davis' self retaining mouth gag with various appliances to hold the tongue depressor, thus doing away with an extra assistant. We saw suction used in very few clinics. All bleeding points are ligated with either silk or linen. In one clinic, double O catgut was used. We saw a very ingenuous tonsil forcep, the Negus, used for ligating.

Various drugs were used in the fauces, either for the relief of post operative pain or prevention of infection, as oil of turpentine, ten per cent protagol, or Whitehead's solution. In the

use of protagol and Whitehead's some of the solution was allowed to flow into the post nasal space as the surgeons thought it prevented infections of the middle ear. No patient is allowed to leave the table until all bleeding has ceased. After the operation, the patient is placed on his right side with his right arm brought back of the body. In some of the clinics, when the patient is returned to his bed, he receives 60 grs. potassium bromide with 20 grs. aspirin per rectum.

On the Continent, most operations on the tonsils in adults are performed under local anesthesia and, on children, a general anesthesia with the patient in the second stage of anesthesia. In the latter they contend it lessens the complication of lung abscess.

Having spent a year in Europe before the war, it was interesting to note the changes that had taken place in operations on nose, ear and throat, especially on disease of the para nasal sinuses and infections of the middle ear. Some of the countries that were formerly very radical, now are very conservative, and those that were conservative, now are more radical, but Conservatism in most clinics has supplanted Radicalism.

In most of the clinics, operations on the nose, including para nasal sinuses, a post nasal plug is inserted through the mouth preceding the operation. In the British Isles, strips of gauze soaked and rung dry in solutions of ten to twenty per cent cocaine with equal parts of adrenalin chloride solution 1-1000 are inserted in the nose one hour before operation. This certainly lessens hemorrhage. We never saw a case of cocaine poisoning while there.

Empyema of the Antrum of Highmore Operation

In very few of the clinics were they radical. In one clinic we saw more Caldwell Luc's performed than in all the others put together. In one of the largest clinics in the British Isles we never saw one, the surgeon in charge informing us that he had not performed one in five years.

The choice of operation in the British Isles is the intra nasal, care being taken to make a very large opening under the inferior turbinate, some removing a small piece of the inferior turbinate with punch forceps and others not disturbing it. In one clinic, the middle third was removed. All kinds of instruments, trocars, punches, chisels and burrs, were used in making the initial opening in the naso antral wall. This was followed

by cutting forceps. In very few clinics, were packing or drainage tubes used.

Note: In a recent paper we called attention to the danger of injury to the teeth in any operation of the antrum through its anterior wall, the only route to safeguard the teeth being through the naso antral wall or hard palate. This we feel can be demonstrated by lantern slides.

In a number of cases the ethmoid cells were curetted but not as a routine. Exenteration of ethmoid cells by Sluder's method was performed in only very few clinics.

Empyema of the Frontal Sinus

In chronic empyema we found the greatest difference of opinion. Most operators sacrifice the naso frontal duct, but a few condemn this procedure. One of our best authorities made the statement that, if naso frontal duct could not be saved, nothing but a complete obliteration of the sinus should be performed. In one case he opened the sinus externally three times before resorting to the radical operation.

The operation of choice was the removal of the floor of the sinus with resection of the frontal process and nasal process of the maxillary and a large rubber tube inserted through the nose and left there from five to fourteen days, then steel dilators were used to keep artificial opening patulous until epidermization has taken place. The removal of the anterior end of the middle turbinate and exenteration of the ethmoid cells, if this has not been done previously, is included in the operation. One surgeon performed the radical operation in a very unique manner. After removing the floor and anterior wall, the patient was placed in the tonsil position, head in occipito atloid position, and, seated at the head of the patient, he completed the operation in the nose and he was able, as it appeared to us, to have a much better view of the field of operation. It certainly was a much easier and quicker method. In one case in which both frontal sinuses had been operated on, that is, removal of the floor and destruction of the naso frontal duct, the patient had to be reoperated. When the surgeon reopened the sinus, he found that he was unable to pass a probe into the nose. Even the bone he had removed from the nose in the previous operation had been replaced with schlerotic bone. In one of the large clinics the surgeon in charge has been using graduated rubber tubes to dilate the naso

frontal duct. He reported seventy cases as cured. I did not mention the intra nasal operation as most of these cases had had this performed previously.

Acute Mastoiditis

As you know, they have two schools over there, one is very strong for early operation and the other believes in waiting until the mastoid abscess has been formed or until it has become localized. It appeared to us that the first school had more followers.

Chronic Suppurative Otitis Media

We saw ionization practiced in only two clinics. Most of the surgeons having given it a fair trial, have abandoned it. In the two clinics where it was used, they were very enthusiastic over it and it was used in practically all cases before resorting to operative procedures.

On the Continent, the greatest change has taken place in the treatment of these cases. We saw very few radicals, but many modified ones performed, the latter consisting of a partial removal of the bridge to its complete removal but, in almost all cases, the contents of the middle ear were not disturbed. Should the case not clear up they contend it was a very easy procedure to complete it into a radical. We were surprised that even in cases with cholesteatoma, a modified radical was first performed. A plastic flap was performed in either method. One of the leading aurists of Vienna in performing either a modified or radical does not stitch the flap or close the posterior wound—just inserts drainage through the external canal and in the mastoid cavity. He says his cases have practically no discomfort and do better than when he stitched the flap and closed the posterior wound. One of the surgeons performs the modified radical by removing the incus and filling the mastoid cavity with tongue flap made from subcutaneous tissues covering the mastoid. He closes the posterior wound without any form of drainage. As to the skin graft, we found just as great a difference of opinion as with us here. In one of the large hospitals they have two services; in one, they never do a skin graft and in the other, in practically every case, we were able to see the end results and they appeared to be equally good. In one hospital, the surgeon removes the posterior membraneous canal in every case. He

contends it only gets in the way. His end results were especially good.

In one of the clinics we saw a very interesting method for skin grafting. The surface from which the skin graft is taken is painted with two per cent methylin blue in absolute alcohol. This is done so you can recognize the skin surface of the graft. Then anhydrous collodion is poured over the surface which is kept taut to prevent wrinkling. After the skin graft has been cut it is placed in salt solution and can be manipulated without its curling up. He cuts the grafts in strips and inserts them through the posterior wound, packing with iodoform gauze through the external canal and closes the posterior wound without drainage. Anhydrous collodion is made by adding collodion in absolute alcohol, one part, and pure ether, two parts. To remove all the water from the alcohol and ether, they are treated with copper sulphate.

Time does not permit us to describe the various methods we had the pleasure of seeing in the treatment of malignant growths in nose and throat; septic thrombosis lateral sinus; brain abscess; meningitis; plastic surgery face; vaccines; Bloc anesthesia; artificial aids to hearing; tuberculosis of the larynx; facial nerve paralysis; operations on the labyrinth; radium in the ear, nose and throat, etc.

In looking back one is discouraged to find that very little progress has been made in the treatment of progressive deafness; ozena; asthma and vaso motor rhinitis.

THE ANATOMY, PHYSIOLOGY AND DISEASES OF THE SALIVARY GLANDS

W. V. MULLIN, M.D.

CLEVELAND CLINIC,

CLEVELAND, OHIO

As one studies the anatomy of the salivary glands, he may derive his information from two sources: the standard descriptive anatomy that gives very definite information as to the size of the glands, their ducts and the exact location of their openings, and the more specialized studies of these structures, such as that made by Churchill Carmalt[1] under the auspices of the George Crocker Special Research Fund at Columbia University.

In his preliminary statement, Carmalt states that he has been impressed with the inaccuracy and incompleteness of the illustrations, especially those of the alveololingual district, which accompany the reading matter in the majority of accepted anatomical text-books. He says they seem to have been handed down unchanged from earlier publications, and in some cases have been obscured by reduction in size. Carmalt's specimens were hardened in formalin and injected, and the illustrations made from them represent accurately the actual conditions encountered in a large series of carefully injected and prepared specimens.

Carmalt divides the adult human salivary apparatus, structurally, into two separate distinct glandular areas: (1) definite major glands with distinct ducts which carry the secretions of each gland to a terminal orifice; for example, the greater submaxillary gland, the sublingual gland, and the parotid gland, and (2) certain areas of the oral, faucial, palatine, pharyngeal and alveobuccal mucous membranes which carry the minute orifices of numerous small separate and distinct glands. This glandular mucosa is distributed over various districts, as for example, the lesser sublingual or alveololingual districts which occupy the lateral areas of the floor of the mouth, the tip of the tongue which contains the glands of Blandin-Nuhn, and the interval between

the submaxillary duct and the lateral surface of the tongue, where are the glands of Weber.

The glands of Blandin-Nuhn are a pair of oval shaped glands on the under side of the tongue near the tip; each gland has one or two excretory ducts opening on the under surface of the tongue. These glands are of the mixed type, having both serous and mucus cells, and they are covered by mucous membrane only. These glands may be congenitally enlarged. They may become cystic and they may contain calculi.

Sublingual Glands—Carmalt divides the sublingual glands into a greater and lesser set, and his injections show that they are not definite in their form or distribution. They have many individual ducts, or some of the lesser sublingual glands may empty into Wharton's duct. They have no capsule and they form an elevation along the floor of the mouth just beneath the mucous membrane. They also are of the mixed type with the mucus cells predominating.

The *parotid gland* is the largest salivary gland. It is purely serous in type. It is yellowish in color and lobulated in form. Carmalt's injections show that in addition to the main gland there are accessory glandular masses which are likely to take the form of a periductal glandular proliferation. Several small lymph glands are usually found embedded on the superficial surface of the gland from which they can be distinguished by their difference in color. They receive afferent lymph vessels from the eyelids, eyebrows, frontal and temporal part of the scalp, the upper part of the cheek, the outer surface of the ear, and the tympanum. Lymphatic drainage from the parotid gland is by way of the deep cervical nodes. The parotid fascia is derived from the deep cervical fascia which forms a capsule for the gland, but as pointed out by Mosher,[2] the inward prolongation of the parotid gland which makes the outer boundary of the pharyngo-maxillary fascia, has no sheath, so that abscesses and tumors of the parotid are unopposed in their spread toward the pharynx. Conversely, the parotid gland may become infected from a retropharyngeal abscess or a peritonsillar abscess that breaks through the superior constrictor muscle. The parotid gland may descend beyond the angle of the mandible and come into close relation with the posterior part of the submaxillary gland, from which it is separated by only a thin layer of the deep cervical fascia, better known as the stylomaxillary or stylomandibular ligament, which runs from the

styloid process to the inner surface of the mandible of the inferior maxilla. The prestyloid portion of the parotid gland is almost in direct contact with the eustachian tube.

The main *submaxillary gland* lies partly beneath the posterior border of the mylohyoid muscle. Carmalt divides this gland into two sections: (1) the main salivary gland, and (2) a group of smaller glandular masses, placed above the mylohyoid muscle, which empty by separate ducts into the main submaxillary duct. These masses are termed accessory or second submaxillary glands and ducts. In his investigations Carmalt has found as many as five accessory submaxillary glands. The submaxillary gland is a mixed type of gland with the serous cell predominating. The gland has a delicate capsule of connective tissue.

The *submaxillary duct* — Wharton's duct — runs from the body of the main submaxillary gland along the floor of the mouth just under the mucous membrane. Its orifice is usually at the summit of a small papilla beside the frenum of the tongue. The walls of the duct are quite inelastic and the duct is accompanied in its course by the lingual nerve. These two factors account for the intense pain which is experienced when Wharton's duct is blocked. It is of great importance to mention that submaxillary lymph glands are embedded in the submaxillary gland, since these lymph glands and their vessels drain a portion of the lip and tongue. The superficial layer of the deep cervical fascia forms the capsule of the submaxillary gland, and as mentioned before, in adults the posterior lobe of the gland is in close proximity to the backward projection of the parotid gland from which it is separated only by a thin layer of fascia as it forms the stylomandibular ligament.

The nerve supply of the salivary glands that controls secretion consists of afferent and efferent fibers from the salivary nucleus in the medulla, afferent and efferent fibers from the chorda tympani, afferent and efferent fibers from Jacobson's nerve, fibers from the glossopharyngeal nerve, and sensory fibers from the fifth nerve. The sympathetic fibers supplying the salivary glands come from the superior cervical ganglion and accompany the external carotid arteries to the glands.

The secretion of saliva is a complex act and involves the cooperation of the epithelial gland cells, blood vessels and efferent and afferent fibers of different cranial nerves, together with a central mechanism by which these are excited to coordinated

activity. Stimulation of any sensory nerve in the body, however, may produce salivary secretion. Ordinarily, between meals the large main salivary glands do not secrete, but the mucous membrane of the mouth is constantly kept moist by the lesser group of mucous glands.

In man, during hunger, the sight, smell or the thought of food causes the secretion of saliva from all the salivary glands. Mastication increases this flow although the total amount of saliva secreted depends upon the nature of the food.

Loevenhart and Hooker[3] reported that when all the salivary glands were removed from a dog, gastric secretion ceased permanently. Fehr, on the other hand, has stated rightfully that extirpation of all the salivary glands is impossible, but that he was able to remove those that secreted by far the greatest amount of saliva, this operation having no appreciable effect on nutrition. The only change he noticed was that the dog drank more water. Since the dog did well, and thrived in every way, his conclusion was that the salivary glands, in the dog at least, were a convenience, rather than a necessity. The injection of saliva into the blood apparently produced no other effect than a temporary fall in blood pressure, but since this result follows the injection of so many substances, this can not be regarded as a specific effect.

During the process of secretion a certain decrease in the rate of flow, or even its cessation, may be caused by stimulation of the afferent nerves. Such an effect might be due either to inhibition of the central secretory center or to constriction of the blood vessels of the gland. This might account for certain inflammatory conditions of the salivary glands that follow surgical operations and certain constitutional diseases.

Rudolph Weber[4] states that one can not speak of normal salivary secretion but that the functioning of each gland is changed by different stimuli. Hydrogen ion concentration tests reveal that after the application of different stimuli the reaction of the saliva varies, rapidly changing from a weak acid to neutral, but only occasionally becoming alkaline.

The viscosity of the saliva is usually in proportion to the percentage of mucin it contains. The same salts may be found in the secretion from all the salivary glands, but the percentage differs in the different glands. The basic salts are sodium, potassium, calcium and magnesium; the acids, hydrochloric, carbonic, phosphoric and sulphuric. Histamine is thought to have a

double action on the submaxillary gland—(1) a secretory effect; and (2) a mechanical effect due to the action of the histamine on the contractile elements of the gland.

Anomalies of the salivary glands have been found in two out of ninety dissected bodies.[5] In one there was complete absence of both parotids, in the other, one submaxillary gland was lacking. Congenital absence of the salivary glands has been found. Periodic swelling of all the salivary glands is usually due to some disturbance of the secretory reflex. Rapid and periodic swelling of the salivary glands may be due to allergy. Such swelling is frequently relieved by an injection of adrenalin. Ranula is a loose term and has no scientific meaning.

The parotid gland is particularly sensitive to the x-rays, even more so than the surrounding skin.

Cysts may form in any of the mucous glands and distention arises as the ducts become blocked. This distention may cause a prolongation of the walls of the gland in any direction; it may burrow through the muscles. These cysts must be differentiated from dermoid cysts, and this is usually not difficult, as salivary cysts are shiny and translucent in appearance, while dermoid cysts are doughy in character. The treatment of salivary cysts is complete removal.

Submaxillary and sublingual inflammation or abscess may lead to Ludwig's angina. The treatment of these conditions is surgical.

A pyogenic infection of the salivary glands and ducts may start as an acute inflammation of the gland. Such an infection has a sudden onset and runs a rapid course. It is usually secondary to some acute or chronic illness such as pneumonia, or typhoid fever, especially in patients of advanced age. It is very apt to follow some surgical operation. In some cases the symptoms will be localized on account of obstruction of the ducts which may be due to edema of the mucosa, to a plug of inspissated mucus from a retarded salivary flow, or to a small calculus. Infection of the salivary glands is more likely to occur during the winter and spring months when acute respiratory infections are prevalent.

The parotid gland is more often the site of postoperative infection and suppuration than either the sublingual or the submaxillary. Several reasons have been advanced for this, but it is questionable whether any of them can be proven. Dyball[6] sug-

gested that at the time of operation some toxin is liberated that selects the parotid gland. Deaver[7] thinks it may be a metastatic process of pyemia, while Bunts[8] observed that postoperative parotitis is more common in patients with poor teeth, gingivitis, oral infection and insalivation. He stated that dryness of the mouth is common after general anesthesia, especially when atropin has been given; and in such conditions the mouth of Stenson's duct, which does not have the advantage of being protected by the tongue as do the ducts of the submaxillary and sublingual glands, is unusually patent, so that infection can readily travel to the glands through the duct or by transmission along the lymphatics around the duct. Loew has suggested that the mucin-containing secretions of the submaxillary and sublingual glands are bactericidal, while the serous secretion of the parotid is not. Whatever theory one may hold, it is practically of value when the patient is receiving no food or fluids by mouth, and when much morphin or atropin is being given, or when he is being fed by gastrostomy, to keep the mouth in a very clean condition, and to stimulate secretion by giving the patient gum to chew at frequent intervals.

Salivary calculus—Calculi of the salivary glands are of not uncommon occurrence, the submaxillary gland more often containing calculi than any of the others, although no plausible reason for this can be given.

It seems to be a fact that all herbiverous animals are subject to salivary calculus, while the carniverous animals are almost free. A calculus weighing 11½ ounces has been removed from the submaxillary gland of a horse. It has been noticed that during fasting new deposits of tartar are not found on the teeth, but when fed with a variety of vegetables, the teeth soon show calcareous deposit near the gingival margin. Therefore, it would appear that a herbiverous diet leads to calcifications in the mouth and the glandular structure.

Badanes[9] calls attention to the importance of the presence of calcium oxalate in salivary calculi. All vegetables, and some fruits, are rich in oxalic acid combined loosely with some organic base. When this oxalate comes in contact with the calcium salts of the saliva, for which it has a great affinity, it is precipitated as an insoluble calcium oxalate, which together with mucin and other salts and food debris, adheres to the surface of the

teeth. By the action of mastication the free surfaces are cleaned automatically but not so well near the gingival margin.

As for *mixed tumors* of the parotid gland—this subject has been thoroughly covered by McFarland.*[10] From a study of ninety tumors of the parotid region, in all of which the postoperative history was traced, he draws the following conclusions:

1. The theory of the "enclavement" (enclosure within a foreign territory) or accidental sequestration of embryonal cells during the early and complicated development of the face and neck affords the most satisfactory explanation of the origin of mixed tumors of the parotid region.

2. Mixed tumors are individual entities, having no relation to the normal structures in which they occur, but from which they do not arise.

3. These tumors are inherently benign but commonly recur after excision, and if frequently disturbed become locally destructive and invasive without causing metastases.

4. The histology of mixed tumors is extremely complex, but on that very account their microscopic diagnosis is usually very easy. On the other hand, the immaturity, atypical arrangement and confused intermingling of the various tissue components easily lead to misinterpretations as to their nature and mistakes as to their dispositions. It follows that histologic variations among mixed tumors have no bearing upon prognosis. Microscopical findings, therefore, except for the fact that they show that the lesion is a mixed tumor, are misleading, rather than enlightening.

5. A malignant change in mixed tumors, whether "sarcomatous" or "carcinomatous" in type, must be rare, and its occurrence is difficult to prove.

6. The rapid enlargement of a mixed tumor of long duration and slow growth is not the result of malignant change.

7. As intervals of ten, twenty, and even thirty years may elapse between the operative removal of a mixed tumor and its recurrence, caution should be exercised in declaring any case to be cured.

Tuberculosis, syphilis, actinomycosis and malignancy may involve any of the salivary glands. The treatment is the same as when these conditions are present elsewhere in the body.

*McFarland—Am. Jr. Medical Science, 1926—Vol. 172, p. 804

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DISCUSSION.

Dr. William Mithoefer, Cincinnati, Ohio: I shall limit my remarks to the practical side of this problem. In other words, I shall speak of the acute and chronic enlargement of the glands in the floor of the mouth referring to the submaxillary and sublingual glands.

You have seen some beautiful slides of the anatomy. There are several anatomical facts that must be borne in mind if we look at this subject from the practical standpoint. First of all, we must remember that the lingual nerve lies in close proximity to the glands of the floor of the mouth. Secondly, we must remember that the fibres of the chorda tympani nerve touch both the submaxillary and sublingual glands. And third and most important, we must remember that there are small lymph nodes that lie around the capsule of the submaxillary gland and around the sublingual gland that has no capsule. These small lymph nodes become enlarged as a result of oral spesis, whether from teeth or tonsils or from an infection in the anterior portion of the mouth—lips, tongue, etc. Enlargement of the lymph nodes lying immediately on the salivary glands will cause collateral oedema of the salivary glands and in consequence a chronic inflammation. When these glands become enlarged you can have various clinical symptoms,—a feeling of fulness in the throat, pain in the ear, glossodynia, a burning sensation in the throat. In fact, many of the vague symptoms in the throat are the result of chronic enlargement of the glands of the floor of the mouth.

How are we to detect the enlargement of the glands? Bimanual palpation is absolutely necessary and I believe it should be a routine measure with all of us to make a bimanual palpation of the floor of the mouth in all patients who have suspicious evidence of throat involvement as a result of infection of nasopharynx, nose above or within the mouth.

This bimanual palpation is made in the following way: the index finger of the left hand, protected with a rubber cot, is placed on the left side of the floor of the mouth, and with the hand on the other side pressure is made at the angle of the jaw and manipulation done. One can often find a marked enlargement of the gland and the patient may experience pain. On the right side the forefinger is used, with the left hand at the angle of the jaw. Very often during the manipulation the patient's symptoms become aggravated, especially the earache.

Clinically, we have seen many cases clear up with proper treatment of the glands of the floor of the mouth. I have in recent years made as an indication for tonsillectomy the presence of a painful and enlarged submaxillary gland with an enlargement also of the lymph nodes overlying the gland. We have seen patients who still had throat symptoms following tonsillectomy who were not relieved until proper treatment was used for the glands of the floor of the mouth. We have seen nasal accessory sinus disease very often associated with this enlargement, and two cases of persistent neuritis of the lingual nerve that cleared up after proper treatment. What is the proper treatment? My experience has shown that the best treatment is massage, and this must be done in a gentle way and patient must be taught how to do it at home. Secondly, we have also seen good results from long exposure to the heat lamp, one hour at a time, and also the administration of atophan.

My message to you is to impress upon you the importance of bimanual palpation on the floor of the mouth as a routine measure.

Dr. T. E. Carmody, Denver, Colorado: I have here some probes which I presented before this society some years ago, graduated and designed after the Bowman probes, and which we find especially useful in those disease conditions where we have constriction of the duct.

Dr. Mullin has told you about the anatomy, as has Dr. Mithoefer, and there is very little left to say about that part of it. What Dr. Mullin told you about the mandible is especially significant if you manipulate the submaxillary gland. He showed you how the posterior part of it drops down below the mylohyoid muscle and sometimes covers it. In the diagnosis of diseases of these glands you will find this anatomical fact very important. In manipulating the parotid gland it is comparatively easy to catch it because you can put your finger up at the external auditory canal and your other finger inside of the mouth and detect whether or not you have a calculus present. The difficulty in locating the calculus with either a probe or an x-ray is sometimes so great that we are at a loss.

Injection is difficult in a normal gland, but sometimes you will find a gland that has been diseased for some time and discharging pus which will retain some opaque substance. We have used lipiodol in one case that showed beautifully; in some other cases it did not show at all. I have a few slides which will illustrate a few of the different conditions that we have in these glands.

Dr. Lyle S. Powell, Lawrence, Kansas: I think we should be very grateful for the excellent presentation of this subject today, especially in view of the fact that, at least in my opinion, the rank and file of our pro-

fession do not give the proper attention to this mass of glandular tissue practically surrounding the upper respiratory tract. I have come to realize in the past few months that a great number of cases that consult us complaining of bad breath and bad taste in the mouth are really chronic infections of these glandular structures. These cases for the most part have gone undiagnosed, or perhaps have had tonsils and adenoids removed and sinus operations performed, with no improvement. I would like to suggest that we give a little more attention to these complex structures that are so intimately connected with the often infected respiratory tract.

DR. W. V. MULLIN, closing: I have just three other thoughts. In cases of periodic swelling of the salivary glands, we have tried injections of adrenalin. The extreme sensitiveness of the parotid gland to the x-ray should be borne in mind. It is more sensitive to the x-ray than is the surrounding skin.

Mixed tumors are mixed tumors all the way through. Even if they recur, they do not have a tendency to become malignant.

There is no reason that I can give as to why one gland becomes involved more than another. For instance, the parotid gland is more likely to become inflamed and suppurate following operative conditions on the gastro-intestinal tract, while the submaxillary gland more often becomes involved by calculus.

DR. MOSHER: Suppose you have a periodic abscess of the submaxillary gland due to stone, how often would you operate?

DR. MULLIN: It would depend upon the size of the stone and where it is. If I could get at the stone and get rid of it, I would try that first.

DR. MOSHER: In most cases of abscess of the submaxillary gland, extirpation is the form of treatment that has given me the best results.

NASOPHARYNGEAL ABSCESS—A REPORT OF 155 CASES

SIDNEY YANKAUER, M. D.

NEW YORK CITY

The lymphoid tissue in the nasopharynx has a definite anatomical arrangement within the structure of the normal adenoid. This anatomical arrangement is maintained when the adenoid persists into adult life and can usually be recognized even when the adenoid structure has become distorted by hypertrophy.

Normally, the adenoid consists of sheets or combs of lymphoid tissue, separated by clefts, and attached to the vault of the nasopharynx along anteroposterior lines. The central cleft begins behind the attachment of the vomer, where it is shallow, and extends backwards in the median line, becoming deeper at its posterior extremity, where it ends in a fossa known as the recessus medius. On each side of the central cleft is a central comb. Outside of each central comb there is a first lateral comb, separated from the central comb by the first lateral cleft and usually there is an incomplete second lateral comb separated from the first by the second lateral cleft. There are, therefore, a central and two lateral clefts, running parallel to each other in an anterposterior direction. These clefts unite posteriorly in the recessus medius. Frequently, the second lateral combs come together posteriorly behind the recessus medius, forming a transverse comb.

In the embryo, the hypophysis cerebri takes its origin from an indentation in the nasopharynx, the location of which is identical with the recessus medius of the adenoid. The indentation grows upward into a prolonged stalk terminating in a mass which becomes the anterior portion of the hypophysis. The stalk itself atrophies but, as in all embryonal structures of this type, remnants of the lumen occasionally remain. Such a remnant is in very rare instances found in the nasopharynx. It consists of a cyst located behind or above the recessus medius. This is the so-

called pharyngeal bursa. It is rarely found, but when present may become the seat of a suppurative process.

In 1885, Tornwaldt, of Danzig, published in the form of a monograph, a series of clinical observations in which he declared that he had seen numerous cases of abcess formation in the region of the recessus medius. These abcesses varied in size from one or two minims to several centimeters, and the contents varied from serum or clear mucus, to creamy pus. Because of the fact that these abcesses are situated in the place where the pharyngeal bursa originated, Tornwaldt concluded that these cases were all cases of pharyngeal bursitis.

His observations were not generally confirmed; first because others did not see such cases nearly as frequently as Tornwaldt did, and secondly, because the presence of an uninflamed pharyngeal bursa was a most rare anatomical find. For a number of years an animated discussion followed the announcement of Tornwaldt. In a general way, the outcome of the discussion was, that because the pharyngeal bursa is a rare anatomical find, therefore, the cases which Tornwaldt saw must be very unusual cases, a conclusion which seemed to be verified by the fact that no others were able to report similar cases, in like number.

This conclusion was erroneous for two reasons: first, because Tornwaldt's claim that these abcesses were due to inflammation of the pharyngeal bursa was unequivocably accepted, and secondly, because the negative findings of others were given equal weight with the positive findings of Tornwaldt. A study of the clinical reports of Tornwaldt's cases has convinced me that the cases which he saw were identical with the cases upon which this report is based, and which, as I will try to prove, can be attributed to another origin than inflammation of the pharyngeal bursa; namely, to an inflammation of the recessus medius of the adenoid. The frequency with which Tornwaldt saw these cases was due to unusual diligence, and skill on his part; for he made it a routine practice in his clinical work to cocainize each one of his patients sufficiently to enable him to introduce a self-retaining palate retractor so as to make possible the examination of the nasopharynx in a convenient and thorough manner.

In the year 1910, the writer devised a direct speculum for the examination of the nasopharynx. With this instrument it is possible with a minimum amount of cocaine, and with a minimum amount of circumstance, to make a thorough examination of

the vault of the nasopharynx. Because the use of this instrument has become a matter of routine practice in my office, I have been able to verify the observations of Tornwaldt not only as to the presence of these abcesses in the nasopharynx, but also as to their frequency; but my observations have convinced me that these abcesses are due to suppuration in the remnants of previously inflamed adenoid tissue and not to inflammation of a supposed pharyngeal bursa. This conclusion is based on the following data:

1. A study of the ages of the patients presenting these abscesses shows that the largest number of cases occurred between the ages of 20 and 30—72 of the 155 cases. The next most frequent decade was from 30 to 40—32 cases. During this period of life, from the 20th to the 40th year, which is the period immediately following the involution of the adenoid mass, two-thirds of the cases occurred.

2. The statistics show that of the 155 cases, only 11 patients stated that they had at any time been operated upon for the removal of adenoids. 93 per cent of the patients, therefore, never had their adenoids removed.

3. If the nasopharynx of adults whose adenoids have not been removed by operation be studied, there will frequently be found in the mucous membrane of the nasopharynx, one or more openings into which a probe may be introduced for a distance of ½cm. Most frequently, a single opening is found occupying the site of the recessus medius of the adenoid. When an additional opening is found it is almost invariably situated in the median line anterior to the above mentioned opening. Sometimes a series of three or four successive openings are found in the median line and the probe shows that they communicate with each other. The bridges of tissue between the openings are fragile and can easily be broken down with a probe, converting the series of openings into a slit corresponding to the median cleft of the adenoid.

4. Where an abscess is present, a small dimple can sometimes be seen on the surface of the swelling. When the abscess is opened with the probe, the opening is converted into a slit. If nothing further is done the parts will soon present exactly the same appearance which is seen in a nasopharynx which presents one of these crypt-like openings but which has never been the seat of an abscess.

5. I have actually observed cases passing from the stage of abscess to the stage of a crypt and reappearing again as an abscess.

6. With one or two exceptions all these abscesses occurred in the median line.

7. It is therefore evident that this condition of abscess formation is one which occurs in adults whose adenoids have never been removed by operation. It occurs shortly after the period when the involution of the adenoid mass has been completed, and in cases in which adhesions between the combs of the adenoid tissue have converted the median cleft into a more or less completely closed cavity.

The history of these cases and the appearance of the nasopharynx corresponds closely with many of the histories and descriptions recorded by Tornwaldt and I am convinced that the cases that I have seen are the same as those which he described but which he erroneously attributed to an inflammatory process in a supposedly persistent pharyngeal bursa. On the other hand, I have seen one or two cases in which a larger cavity appeared which could not be entered with the probe but required a knife, which contained a larger amount of serous fluid and which were considered to be true cases of pharyngeal bursitis.

Diagnosis:

A study of the statistics of this series of cases shows that there is a preponderance of females, there being 90 females and 65 males.

The ages vary from 5 years to 65 years with most of the cases between the ages of 20 and 40.

It is of some interest to note that the majority of the cases occurred either in the late spring, from March to June inclusive, or in the late fall, from October to January inclusive. This seasonal distribution is probably associated with weather conditions, and might be expected to vary with the climate.

It is also of interest to note the frequency with which these cases occurred in relation to other nasal and nasopharyngeal conditions. The total number of cases was collected over a period of years. But of 760 patients of all kinds of nose, throat, laryngeal and endo-bronchial diseases seen in private practice during the year 1928, there were 28 cases, or 3.8 per cent with abscesses of this kind. During the first four months of this year, of 315

patients of all kinds there were 13 nasopharyngeal abscesses, or 4.1 per cent. This slight increase in the present year is due to the fact that it includes the months in which the disease is most common and that during this period there was an epidemic of upper respiratory infections.

The local diagnosis depends upon the use of the direct nasopharyngeal speculum. When a reflecting instrument is used to examine the nasopharynx the light rays are projected upon the pharyngeal mucosa from a reflecting surface which is placed far back near the posterior pharyngeal wall. The angle of incidence of the rays as they strike the surface of the nasopharyngeal mucous membrane is greater than the angle of total reflection of the mucus which covers the surface. In like manner, any light rays which may emerge from the surface of the mucous membrane is totally reflected at the surface of the superficial layer of mucus. Therefore, unless the nasopharynx has been entirely freed of mucus and maintained free of mucus during the examination, all that will be seen in a reflecting instrument will be a uniform pinkish sheen with the details of the texture of the surface obliterated. In the majority of cases it is practically impossible to recognize the contour of the nasopharynx which is essential for the diagnosis of the condition. On the other hand, with a direct speculum the presence of a small quantity of mucus does not interfere with the visibility of the surface of the mucous membrane and, besides, the surface can easily be kept free of mucus with suction.

An abscess is characterized by the presence of a swelling in the region of the recessus medius, but the swelling may not manifest itself as a rounded protuberance but merely as an elevation of the concavity of the vault of the nasopharynx, a flattening out of the concave surface. When the swelling can be recognized, it may be red, smooth and glistening or it may present a dull grayish, almost polypoid appearance. Occasionally a dimple is found on the surface of the swelling. Not every such swelling however, contains pus, and the diagnosis is not complete until the swelling has been opened and its contents noted. In all cases except one or two it has been possible to penetrate the swelling with a probe, whereupon pus was found to exude alongside of the probe. The puncture wound made by the probe may then be converted into a slit, and the contents of the cavity completely evacuated. It was in this way that each one of the 155

cases upon which the present article is based, was diagnosed, and it was only after the fluid contents of the cavity were actually seen that the diagnosis was considered complete. Of the 155 cases, the character of the contents was noted in 142. In 121 or 86 per cent of these, the contents were recorded as thick, creamy pus. In five of these, exuberant granulations could be seen protruding from the wound after the pus was evacuated. In the remaining 21 cases or 14 per cent, clear, thick, glairy mucus was evacuated.

Unless and until the rhinologist acquires a familiarity with the appearance of the nasopharynx in the direct nasopharyngeal speculum, and as long as he depends entirely upon reflecting instruments for the examination of these parts, it will be impossible for him to recognize the presence of these abscesses in all of the cases and it will be impossible for him to treat them in an efficient surgical manner.

In practically all of the patients the abscess in the nasopharynx was not the only pathological condition present, nor was it seemingly the most important. The associated conditions of the nose and throat include practically all of the diseases generally found there. In over one-third of the cases, diseased tonsils were associated with the nasopharyngeal condition and deviated septa and accessory sinus disease were not uncommon.

A few cases of posterior cervical adenitis seemed to be directly associated with the presence of these abscesses.

As already indicated above, the abscesses had a tendency to open spontaneously and discharge their contents, and then to close without indicating the fact by any evidence of an acute process. Under these circumstances, it is manifestly impossible to distinguish the symptoms caused by the presence of such a small abscess from those caused by the associated diseases. Nevertheless, by reviewing the figures obtained from a study of these 155 cases, it was possible to obtain some significant data.

For this purpose, the symptoms presented by the patients were separated into groups. The first group are those symptoms which might reasonably be attributed to the presence of an open abscess cavity discharging its contents into the nasopharynx. This group included such symptoms as nasal discharge, postnasal discharge, and frequent colds including hoarseness, foul taste or odor, nausea, hawking and coughing, attacks of sneezing. The

total number of patients presenting these symptoms was 103 or 66½ per cent.

The second group of symptoms are those which might reasonably be attributed to a closed abscess cavity in the nasopharynx. This group includes headache and pains in various parts of the head and neck including a number of cases diagnosed as sphenopalatine neuralgia, nasal obstruction, sore throat, nasal speech, cervical adenitis. The number of cases in this group was 108, or 68 per cent. It is therefore evident that about two-thirds of the patients complained of symptoms attributable to an open abscess in the nasopharynx, and about two-thirds of the patients complained of symptoms due to a closed abscess. These figures can only be reconciled by assuming that approximately one-half of all the patients complained of symptoms when the cavity was closed as well as of symptoms when the cavity was open; that of the remainder, some complained of symptoms only when the cavity was open, and some only when the cavity was closed.

The headaches of which the patients complained included all kinds of pain in the head and neck but a number of patients, perhaps one-half of those complaining of headache, described the headache in a peculiar way. The headache is felt at the back of the head and resembles the pain of sphenoid sinusitis. However, when a patient with sphenoid sinusitis is asked to point out where his headache is situated, he will place his entire hand upon his occiput; but when a patient who has a headache due to one of these abscesses tries to point out its location, he will place one finger on the nape of the neck just below the occipital bone—a point which corresponds closely to the location of the abscess. In other words, the headache due to nasopharyngeal abscess seems to be much more localized than the headache of sphenoid sinusitis. Although this particular type of headache occurred in only a minority of all the patients, when it did occur, an abscess was invariably found in the nasopharynx, and the opening of the abscess was invariably followed by the disappearance of the headache. In some instances, these headaches were severe and many unsuccessful attempts to relieve them had been made. It is the only symptom of these abscesses which seems to me to be so definitely and clearly associated with the presence of the abscess that it could be regarded as pathognomonic.

The next group of symptoms are those relating to the ear and it is very surprising that in a suppurative disease of the

nasopharynx there were only 23 or about 15 per cent of the patients who complained of any ear symptoms whatsoever. Only one-third of these, or 5 per cent, at any time had a suppurative otitis either acute or chronic. The other ear symptoms were vertigo, tinnitus, earache and deafness.

In like manner it is perhaps equally surprising that there were comparatively few, not more than 5 per cent of the total, who complained of focal symptoms such as nephritis, rheumatism, persistent fever, etc. Nevertheless, in some of these cases the relief of rheumatism followed as promptly as it sometimes does after the removal of tonsils and there were three cases of persistent slight rise of temperature which were relieved by the removal of such abscesses.

There were a large number of nervous symptoms whose relation to the abscesses may be obscure; they included neurasthenia, insomnia, reflexes of various kinds, and aches and pains in various parts of the body. One case of frequent micturition was immediately relieved after opening such an abscess.

A number of instances occurred in which a diagnosis of accessory sinus disease had been made and for which various sinuses had been operated upon. In spite of the fact that the intranasal appearances seemed to indicate that the sinuses were healed, nasal discharge and headache persisted and were relieved only when the abscess in the nasopharynx was opened.

The number of cases seen in hospital and dispensary practice was very much smaller than those seen in the writer's private practice but this fact itself is significant. It is probably due to the circumstances that the writer's practice is entirely a consultant practice and that the patients that are brought to him are those in whom careful diagnosis and skillful treatment failed to relieve the patients. In other words, it is among the unsuccessful cases of the average rhinologist that these cases have been found. This fact should make the recognition and treatment of these nasopharyngeal abscesses a matter of great interest and importance to all rhinologists, for in the writer's practice, one of these abscesses was found in every 25 patients. It is certain that a pathological condition which occurs in the practice of a single rhinologist every ten days cannot be dismissed as a rare or unusual or unimportant disease. If this figure truly represents the proportion of undiscovered nasopharyngeal abscesses among the unsuccessful cases of other rhinologists, a better knowledge

of the diagnosis and treatment of this condition will add a measureable degree to the theraupetic efficiency, and therefore to the professional prestige of our specialty.

The treatment of these cases, as has already been suggested above, is carried out through the direct nasopharyngeal speculum. The cocainization necessary for the introduction of this instrument is as follows:

Two applicators are wound with absorbent cotton for a distance of 1½ cms. from the end; the tuft of cotton should not be more than 4 mm. in diameter and the end rounded off smoothly. About 2 cm. of the end of the applicator are bent to a shape corresponding to the shape of a eustachian catheter. These applicators are saturated (but not dripping) with from 5 per cent to 20 per cent cocaine solution with adrenalin and introduced through the nostril with the bent part downward so that the bent part comes to lie on the upper surface of the soft palate. They are left in situ for ten minutes. The instrument is introduced in the usual manner until the swelling in the nasopharynx is brought into its lumen. The use of suction may be necessary to keep the field clear of secretion especially if there is nasal discharge from an associated sinusitis. A stiff steel probe is applied to the swelling. It can easily be thrust through the walls of the abscess cavity. A small quantity of secretion usually exudes alongside of the probe. The probe is then forcibly moved downwards and backwards, converting the puncture wound into a slit and evacuating the contents of the cavity. There is usually brisk, but temporary hemorrhage. When this has subsided an application of 20 per cent solution of nitrate of silver may be made to the interior of the cavity. In many cases this is sufficient to eliminate the disease. The lateral walls of the cavity separate, the recessus medius becomes shallow and the surface of the nasopharynx smooth. In a number of instances, however, the cavity reforms. This is due to the fact that the walls of the cavity, which are composed of the central combs of the adenoid, still contain a considerable amount of lymphoid tissue. They remain thick and rounded and maintain the original formation of the central cleft and recessus medius. The removal of this remaining lymphoid tissue is therefore indicated. This can be accomplished without difficulty through the direct speculum, using for this purpose an ordinary punch forceps of sufficient length to reach through the speculum. Occasionally the amount of lymphoid tissue is un-

expectedly large and a more complete operative procedure is necessary. The removal of this tissue then becomes equivalent to the removal of adenoids from the nasopharynx of the adult, and may be accomplished by a procedure which serves this purpose. The writer has successfully used the method taught by Dr. Joseph C. Beck, which consists of introducing rubber cords through the nares, bringing them out of the mouth and using them as palate retractors, and in this way curetting the nasopharynx under direct vision.

Not the least important feature in connection with these abscesses is the attitude which rhinologists have assumed towards this condition. Although at one time it was the subject of animated discussion under the name Tornwaldt's disease, in late years it seems to have been omitted almost entirely from consideration and to have become lost to rhinologists as a factor of any importance in the pathology of the nose and throat. Not only do we find discussion of the disease almost entirely absent from the literature but in the text books published in the English language during the last ten years, half of them do not mention the existence of such a condition at all. The other half of the text books describe under the name of Tornwaldt's disease, an inflammation of the pharyngeal bursa. Inflammation of the pharyngeal bursa is an entirely different pathological condition as the bursa is situated behind the mucous membrane of the nasopharynx and behind and above the recessus medius. The contents of the pharyngeal bursa are more often clear serous fluid or glairy mucus. When the bursa is the seat of suppurative inflammation, the differential diagnosis from suppuration in the recessus medius is difficult, but the writer believes that a distinction can be made by the surgical procedure necessary for the opening of the abscess. The recessus medius being situated entirely in the remnant of adenoid tissue, the abscess cavity can be entered easily by means of a probe and the cavity opened in this way. It is practically never necessary to use a knife. On the other hand, when the cavity to be opened is a pharyngeal bursa, the probe cannot be made to enter the cavity. The mucous membrane of the nasopharynx is too resistant for this simple procedure. It becomes necessary to use a knife.

In those text books which have described Tornwaldt's disease it is always pharyngeal bursitis which is described. I have found only one exception to this rule. In the first edition of

Ballenger's text book on Diseases of the Nose and Throat, Tornwaldt's disease is described definitely as an inflammation of the recessus medius. In the year 1912, a book on Diseases of the Nasopharynx, by Adair Dighton, was published in England. Dighton accepts the description of Tornwaldt's disease published by Ballenger and repeats the description almost verbatim. The same description is repeated in the later editions of Ballenger. With this exception, there is no publication that I have seen in which Tornwaldt's disease is described as an inflammation of the recessus medius. I have found no other text book in which inflammation of the recessus medius as such, is described at all. The disease has practically been lost to rhinologists and the only excuse which the writer has for attempting to resurrect this lost disease and to save it from oblivion is the fact that never a fortnight passes in which he does not see at least one case and that the relief of a number of obstinate cases which has been accomplished by the discovery and cure of these abscesses have been among the most satisfactory cases in the writer's experience.

DISCUSSION.

Dr. Joseph C. Beck, Chicago: The author has presented his paper in such a brief manner that he evidently thinks we all have read the Presession Volume. I think probably a good many have not read it, and I will admit that I only read it because I knew I was going to be called upon to discuss it.

On most of the points I agree with Dr. Yankauer; on some I do not. In the first place, the statement that textbooks do not mention this condition. I am sorry, although he mentioned my name in the paper in connection with drawing the palate forward, that he did not mention that my book on Pathology had this condition differentiated. Dr. Yankauer has the two conditions very clearly differentiated—one is an external disease of the nasopharynx, and the other is a bursa not open to view. But these bursae do become infected. Most of the cases he reports are those of the open type of infection in which he find definite healing over of the pockets, so to speak, in which the treatment is followed by slitting the retention pocket, with remarkable results both local and general.

Many conditions are mentioned in the paper, such as systemic infection from the tonsils.

I think if you will read this paper you will get a good deal out of it. The point the doctor stresses is the importance of routine examination of the postnasal spaces. It is like examination of the larnyx, so often it is not done in a routine nasopharyngeal examination, and while the larnyx is easily examined, I am sure that with the excellent instruments the Doctor has designed that can be used without first anaesthetizing the nasopharynx, it should be done routinely.

I thank him for referring to the method that I have advocated of using the rubber tubes, because I feel one can make a better and clearer diagnosis and get a better exposure by this method than by his own instruments.

Dr. Samuel Salinger, Chicago: I must confess a considerable elation upon reading Dr. Yankauer's paper, inasmuch as it happens to coincide with several experiences I have had in studying this disease. At the same time I feel that he has disclosed a secret which has been the means of my acquiring several patients as well as considerable emolument, due to the fact that the diagnosis in some of these cases had been completely missed.

A few years ago I had occasion within a few weeks to see several cases of acute infected adenoids with unexplained pain in the ear. I found some temperature and suboccipital pain, swollen adenoid tissue with pus exuding from the lacunae. I found a very good description of the disease in Heyman's Handbuch, and also a number of references in the literature on the subject. I was amazed to find that enlarged and infected adenoids had been reported up to the age of seventy.

I have a slight criticism to make and that is with reference to the title of the paper. It is a little misleading. The disease that Dr. Yankauer describes is limited solely to the adenoids, whereas nasopharyngeal abscess might be another condition. It might be a descending suppuration from the petrous bone, or it might be an abscess of the suprapalatal retropharyngeal glands. I think probably the term "Empyema of the Adenoid Recess," or "Acute and Chronic Infections of the Adenoids" might be more explanatory. I am sure, however, that if after this meeting we will read the paper over carefully and follow the valuable suggestions, Dr. Yankauer's consulting practice is likely to suffer.

Dr. Sidney Yankauer, closing: I think the most important thing that has been brought out by the discussion is that you have failed to read the Presession reprints. I think it is important to stress that, because if the writers of papers are going to have their papers ready by June first, and if the purpose of the Presession Volume is to save time at the meetings, I think everybody should read the Presession reprints.

SYMPOSIUM ON THE ETHMOID.

THE SURGICAL ANATOMY OF THE ETHMOIDAL LABYRINTH

HARRIS P. MOSHER, M. D.,

BOSTON, MASS.

The subject which is assigned to me, and which begins this symposium, namely the Surgical Anatomy of the Ethmoid, suggests to my mind almost automatically the following topics. The approach to the naso-frontal duct, the removal of the floor of the frontal sinus from the orbit, the presence of a fronto ethmoidal cell, the relationship of the posterior ethmoidal cells to the sphenoidal sinus and the optic nerve, and, finally, the partial or the complete exenteration of the ethmoidal cells.

The surgical anatomy of the ethmoidal labyrinth means the applied anatomy. Therefore, a detailed description of the anatomy is called for only where it has a surgical application. *(Fig. 1.)*

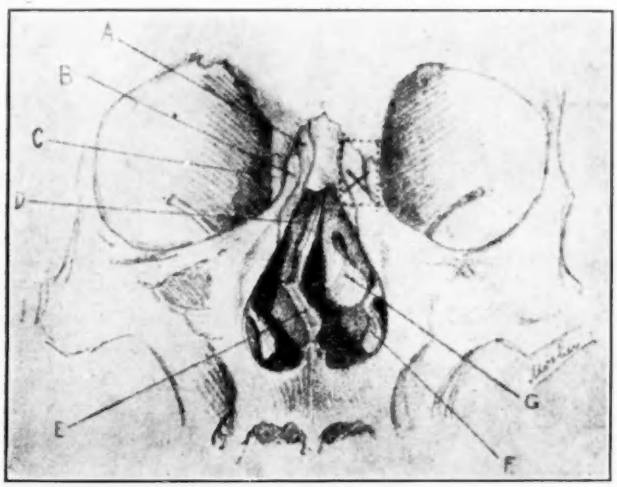


FIG. 1—The Relationship of the anterior end of the ethmoidal labyrinth to the middle turbinate, the tip of the ascending process of the superior maxilla, and the lacrimal bone. The position of the labyrinth is shown by the dotted lines; the cross shows the level at which the curette enters the labyrinth. A, nasal bone; B, ascending process of superior maxilla; C, lacrimal bone; D, middle turbinate; E, septum—marked deviation to right; F, inferior turbinate; G, middle turbinate, much enlarged.

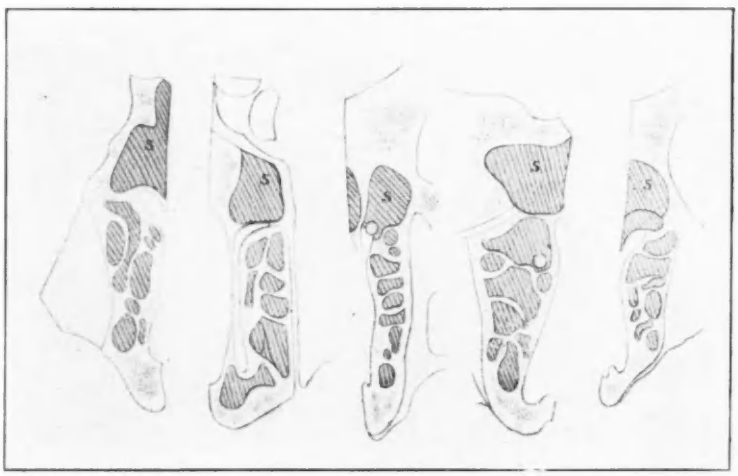


FIG. 2—Specimens of the ethmoidal labyrinth cut horizontally. The specimens show the variations in the form of the labyrinth. Each labyrinth (about three fourths of the distance back) tends to bulge toward the septum. This inward bulging makes it difficult to see the inner or nasal face of the sphenoidal sinus. In order to do away with this bulging portion of the labyrinth the superior turbinate must be removed flush with the anterior face of the sphenoidal sinus. The third specimen is a narrow labyrinth and the fourth a very wide one.

If the ethmoidal labyrinth was placed in any other part of the body it would be an insignificant and harmless collection of bony cells. Placed where Nature put it, it has a number of major relationships so that the diseases and surgery of the labyrinth often lead to tragedy. It has been said that the ethmoidal operation is the easiest in surgery. So it is to the operator who lacks a surgical conscience. Theoretically, the operation is easy. In practice, however, it has proved to be one of the easiest operations with which to kill a patient. *(Fig. 2.)*

The lateral mass of the ethmoidal labyrinth is a thin, bony box which is roughly shaped like a pyramid. The wider part, or the base, is posterior, the blunt apex is anterior. The superior face of the box is wider than the inferior.

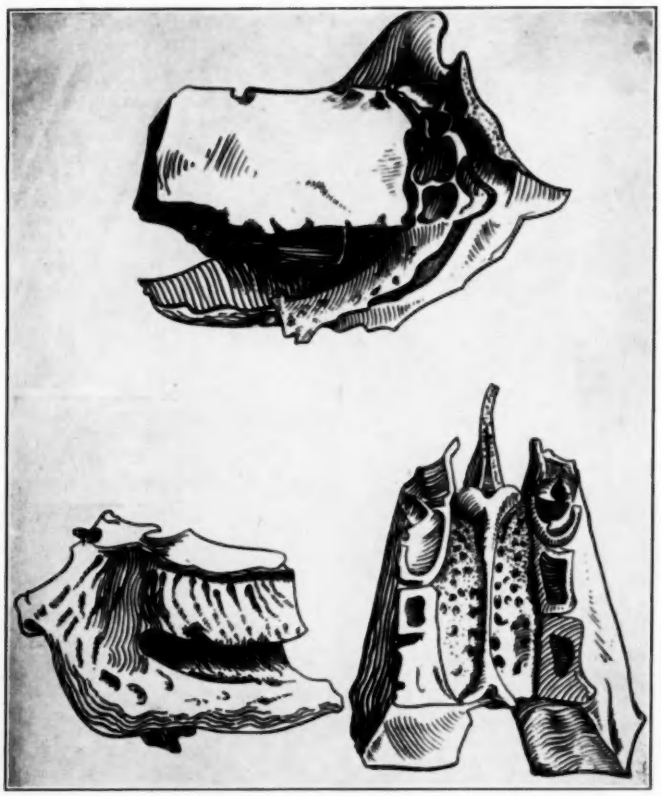


FIG. 3.—The Ethmoidal Bone.

The ethmoidal labyrinth, or rather the ethmoidal pyramid, occurs in two forms, the typical pyramidal form, and the flat thin form in which the anterior and the posterior diameters are the same and both are much less than normal *(Figs. 2-3.)* One expects in exenterating the ethmoidal cells to have more space posteriorly than anteriorly. This is true in the usual type of pyramidal labyrinth. When, however, the labyrinth is of the thin type, and of the same width throughout, the operating space does not increase. In small thin heads the possibility of this formation must be borne in mind.

The anterior posterior length of the labyrinth is 4 to 5cm, the vertical height 2½ to 3cm, the width of the labyrinth posteriorly, that is, the width of the base, is one centimeter and a half or 1.5. The width of the labyrinth anteriorly is one half centimeter or (5mm).

If all the partitions within the labyrinth are broken down so that it becomes literally a bony box, it will hold from 8 to 10cc (3 drachms).

The lateral mass of the disarticulated ethmoidal bone is incomplete in three places. First, the superior surface along its inner edge has a row of incomplete cells which are made complete by a similar row of half cells lining the ethmoidal notch of the frontal bone. The anterior third of the orbital surface of the lateral mass has a series of uncapped cells which are made complete by the lachrymal bone. The posterior surface, or the base of the labyrinth, has a common wall with the outer half of the front face of the corresponding sphenoidal sinus. Thus in the disarticulated ethmoidal bone, if the lateral mass happens to be complete posteriorly, the outer half of the front face of the sphenoidal sinus of the corresponding side is wanting. Both cannot be complete because they have a common wall.

The Major Relationships

The roof of the ethmoidal labyrinth is a continuation of the floor of the anterior fossa of the skull. The relationship of the labyrinth superiorly with the anterior fossa and its contents is the most important. The superior surface is the tragedy surface. It is continuous internally with the cribriform plate and rises in a dome above it some two or three mm. The outer surface of the labyrinth makes the inner wall of the orbit.

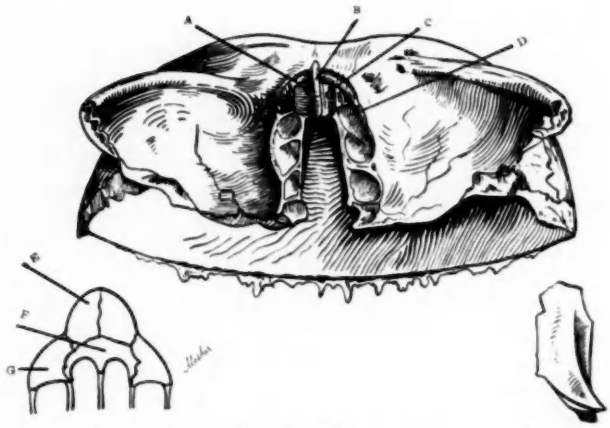
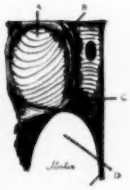


FIG. 4—Under-surface of the frontal bone and a cross-section of the nasal bones.
A. Nasal notch. B. Nasal spine. C. Roof of nasal cavity. D. Naso-frontal duct. E. Nasal bone. F. Nasal notch frontal bone. G. Ascending process of superior maxilla.

FIG. 5.—Drawing from a specimen in which the whole ethmoidal labyrinth has been exenterated. The posterior third of the superior turbinate has not been removed. This bulges inward and obscures the inner or intranasal part of the front face of the sphenoidal sinus. It is necessary for correct orientation in operating to remove the superior turbinate flush with the front face of the sinus. (See Fig. No. 6.)
A. Outer or ethmoidal face of the sphenoid. B. Attachment of superior turbinate. C. Inner or nasal surface of face of sphenoid. D. Os planum.

FIG. 6.—Diagrammatic drawing of the front face of the sphenoidal sinus. When the exenteration of the ethmoidal labyrinth has been completed and before the sphenoidal sinus is entered the operative field should present this picture. The upper outer posterior angle of the ethmoidal face is the most dangerous area in the operation, i. e., the region of the upper left hand marker A. A, outer or ethmoidal half of frontal face of sphenoid; B, attachment of superior turbinate; C, inner or nasal surface of sphenoid; D, right choana.



Two thirds of this surface are made by the os planum. The anterior third is made by the lachrymal bone. The outer surface of the labyrinth, therefore, is in relationship with the lachrymal bone and the lachrymal sac. That is, the anterior ethmoidal cells are in relation with these two structures and the orbit. The posterior half of the labyrinth contains the posterior ethmoidal cells. These have an extensive relationship with the orbit and at times the most posterior of the ethmoidal cells has a close relationship with the optic nerve. *(Fig. 5.)* The posterior face of the labyrinth, as has just been said, has a common wall with the front face of the corresponding sphenoidal sinus. This means, of course, that the sphenoidal sinus can be entered at this point from the ethmoidal labyrinth. The perpendicular attachment of the superior turbinate marks the inner limit of the common wall on the front face of the sphenoid. The attachment of the superior turbinate roughly divides the front face of the sphenoid into thirds. The outer two thirds are taken up by the common wall. The inner third, which is the space between the attachment of the turbinate and the nasal septum presents as a free surface in the posterior superior part of the nasal cavity. The lower limit of this space is the upper rim of the choana of the same side. So that the choana and the nasal septum act as surgical guides to this part of the front face of the sphenoid. The superior boundary of the nasal face of the sphenoid is the cribriform plate. The nasal face of the front face of the sphenoid contains in its upper part the ostium of the sphenoidal sinus and in its lower part it is crossed horizontally by the spheno-palatine artery which gains the nasal cavity from the spheno-palatine foramen. *(Fig. 6.)*

In the normal pyramidal labyrinth of good size, the lower surface of the lateral mass of the sphenoid overhangs the antrum in what is called the ethmoidal overhang. To summarize, the main relationships of the labyrinth are with the brain above, the orbit and the optic nerve on the outside, the outer part of the front face of the sphenoid posteriorly and the antrum below. The cribriform plate is to the inside of the ethmoidal labyrinth and as long as the operator's instrument remains within the labyrinth it is not in danger.

The anterior boundary of the ethmoidal labyrinth is the posterior surface of the ascending process of the superior maxilla. It is the only boundary which is strong. *(Fig. 7.)* Against this the operator can curet to his heart's content. All the other

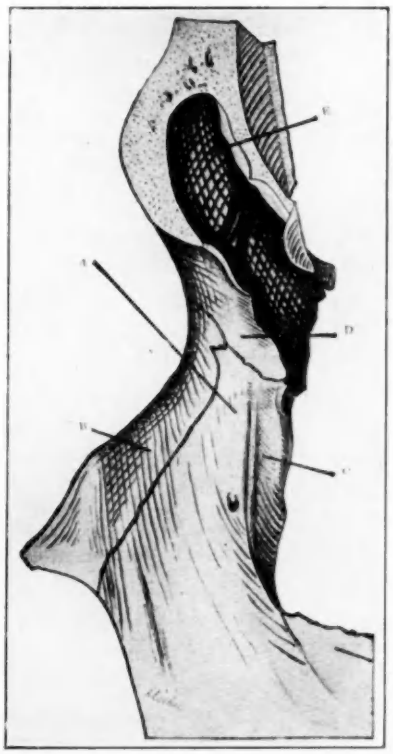


FIG. 7.—Drawing from a specimen to show the relation of the internal angular process of the frontal bone to the summit of the ascending process of the superior maxilla.

A. Ascending process of superior maxilla. B. Nasal bone. C. Lacrimal groove. D. Internal angular process of frontal bone. E. Frontal sinus.

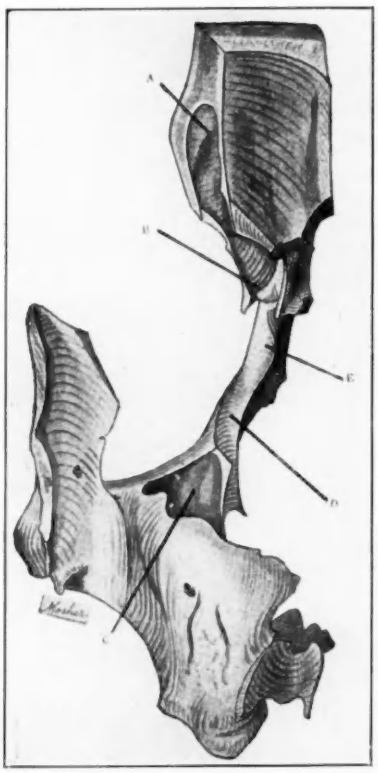


FIG. 8.—Drawing from a specimen which shows how the internal angular process of the frontal bone makes the anterior half of the opening of the naso-frontal duct and overlaps like a shingle the posterior surface of the ascending process of the superior maxilla. A probe, carried upward along the posterior surface of the ascending process of the superior maxilla is guided by it into the frontal sinus. If the frontal sinus is approached from the orbit the surest way to find the sinus is to remove the lacrimal bone and then carry a probe upward along the posterior surface of the ascending process of the superior maxilla. In like manner when the frontal sinus is approached from the nose, the operator first turns the anterior cells into one cavity and then in the anterior part of this locates the posterior surface of the ascending process of the superior maxilla and passes a suitably bent probe upward on this as a guide.

A. Frontal sinus. B. Internal angular process of frontal. C. Antrum. D. Lacrimal groove. E. Posterior surface of ascending process of superior maxilla.

walls of the labyrinth are thin and must be approached with great care. *(Fig. 8.)*

The ethmoidal lamella from which the middle turbinate springs, runs outward to the os planum and upward to the roof of the labyrinth dividing it into two parts and ends at the anterior ethmoidal foramen. All cells in front of the attachment of the basal lamella of the middle turbinate are called anterior cells, are relatively small and average 7 or 8. All cells posterior to the attachment of the os planum are classed as posterior.

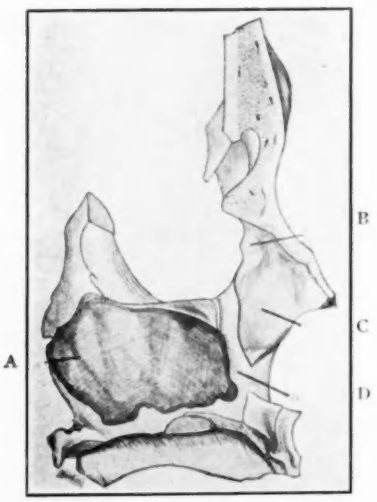


FIG. 9.—Drawings from a specimen to show the ascending process of the superior maxilla, the agger nasi, and the lacrimal groove.

A. Antrum. B. Agger nasi. C. Ascending process of superior maxilla. D. Lacrimal groove.

They are larger than the anterior and average 3 or 4. It is not uncommon to have two large cells make up the whole of the posterior half of the labyrinth. The smallest total number of ethmoidal cells as given by Schaeffer is 3, the largest 15.

The upper half of the outer wall of the nose is made by the ethmoidal labyrinth. The lower half by the base of the ant-

FIG. 10.—Cast from a wet specimen in which the anterior and posterior ethmoidal cells have been exentrated leaving the attachment of the middle turbinate.

rum. From the nose the most accessible part of the labyrinth surgically, is the agger nasi cell, if present. If not, the ethmoidal bulla.

At times the unciform process which is placed in front of the bulla is hypertrophied. When this happens the unciform process would be the most accessible part of the labyrinth.

The lamella of the attachment of the middle turbinate although thin, can be recognized with the curette. The author has met a few instances in the living and a few more on the cadaver in which it was so thick that no force which would be justifiable to use in operating would carry the curette through it. *(Fig. 10.)*

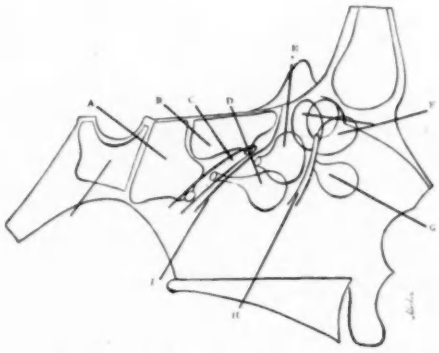


FIG. 11.—Diagram of the arrangement of the ethmoidal cells.

A. Posterior ethmoidal cell. B. Posterior ethmoidal cell. C. Third meatus. D. Lower cell of ethmoidal bulla. E. Upper cell of ethmoidal bulla. F. Cells at upper part of unciform groove. G. Agger nasi cell. H. Unciform groove. I. Attachment of middle turbinate. J. Sphenoidal sinus.

All the turbinates of the nose with the exception of the inferior, or maxillo-facial, spring from the ethmoidal labyrinth. The chief ethmoidal turbinate is the middle. It is equally normal to have one or two ethmoidal turbinates above the middle turbinate. *(Figs. 11-12.)*

The anterior ethmoidal cells are found in the middle meatus in its upper part clustered around the unciform groove and opening into it. The unciform groove is an embryological slit between the unciform process of the ethmoid and the ethmoidal bulla. It usually ends blindly or in an anterior ethmoidal cell. Only in a small percentage of cases is it continuous with the naso-frontal

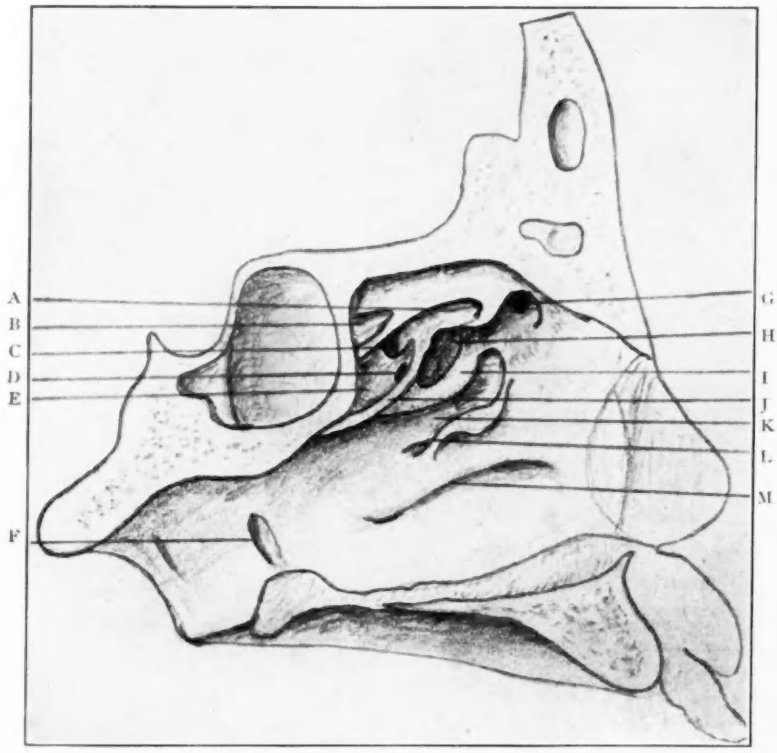


FIG. 12.—Student drawing from wet specimen. The unciform groove ends blindly. The opening of the frontal sinus is above the end of the unciform groove and in line with it.

A. Part of superior meatus. B. Supreme meatus. C. Posterior ethm. cell ostium. D. Posterior ethm. cell ostium. E. Posterior part of superior meatus. F. Eustachian orifice. G. Ostium of naso-frontal duct. H. Groove of bulla. I. Ethmoidal bulla. J. Attachment of middle turbinate. K. Unciform groove. L. Unciform process. M. Atrophied inferior turbinate.

duct. When the groove ends blindly or in an ethmoidal cell, the lower opening of the naso-frontal duct is found in the outer wall of the middle meatus in line with the unciform groove and above it, or as Schaeffer says, contiguous with it but not continuous. Anatomically it is not possible to catherize the naso-frontal duct. As long as gravity holds, however, secretions from the naso-frontal duct readily flow into the unciform groove. Under cover of the flaring lower end of the unciform process the ostium

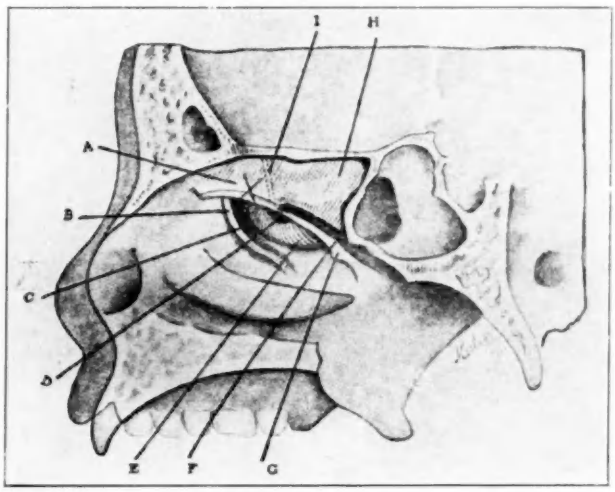


FIG. 13.—Drawing to show the anatomy of the nose after the middle turbinate has been removed in the usual manner. It is important to notice that the upper anterior part of the middle turbinate is still in place, and that this portion of the turbinate bridges over the upper part of the unciform groove. Removing the middle turbinate in this manner neither completely uncovers the anterior half of the ethmoidal labyrinth, nor opens it. The cross indicates the point at which the curet is carried into the labyrinth.

A. Upper anterior part of middle turbinate. B. Unciform process. C. Unciform groove. D. Groove of ethmoidal bulla. E. Ethmoidal bulla. F. Third Meatus. G. Posterior attachment of middle turbinate. H. Superior turbinate. I. Upper part of attachment of middle turbinate.

of the antrum opens into the unciform groove at a right angle. It is difficult, if not impossible, therefore, in the majority of cases, to cautherize the ostium of the antrum. From the surgical standpoint, however, this makes but little difference because it is easy to break into the antrum near the ostium through the membraneous area of the antrum in which the ostium is placed. *(Fig. 13.)*

The writer likes to divide the anterior half of the middle turbinate into an upper part called the superior overhang and a lower

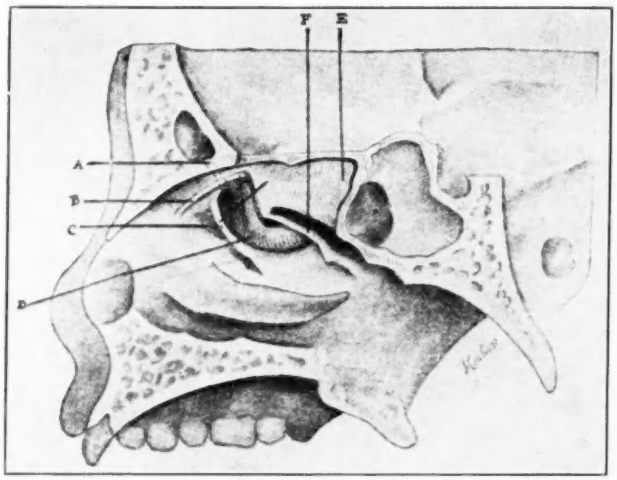


FIG. 14.—Drawing from a specimen in which the middle turbinate has been completely removed. By the removal of the upper overhang of the middle turbinate the anterior half of the labyrinth has been fully exposed. Compare this figure with the preceding one, in which the upper overhang of the middle turbinate is still in place. The usual operative procedure deals only with the lower overhang of the middle turbinate. The upper overhang is on a line with the superior turbinate and for practical purposes, continuous with it. A, naso-frontal duct; B, attachment of middle turbinate; C, unciform process; D, ethmoidal bulla; E, superior turbinate; F, attachment of middle turbinate.

part called the inferior overhang. The point of division is a horizontal line drawn forward from the upper end of the third meatus. The anterior part of the middle turbinate bridges across the unciform groove to gain an attachment to the inner surface of the ascending process of the superior maxilla. On the inner surface of the ascending process there is a ridge called the agger nasi. At this point the ethmoidal cell, known as the agger nasi cell, is developed. The lower overhang of the middle turbinate is known in every day surgical parlance as the tip. *(Fig. 14)* It serves to cover the lower part of the bulla and the lower part of the unciform groove. It is below the region of the naso-frontal duct. Secretions from the frontal sinus and the ethmoidal cells drain into the unciform groove, and from here overflow into the anterior part of the nose or make their way down into the antrum. Secretions from the posterior ethmoidal cells flow backward, by way of the third and fourth meatuses, gain the sphenoidal ethmoidal recess between the posterior end of the ethmoidal labyrinth and the front face of the sphenoid and appear on the posterior wall of the pharynx or on the upper border of the

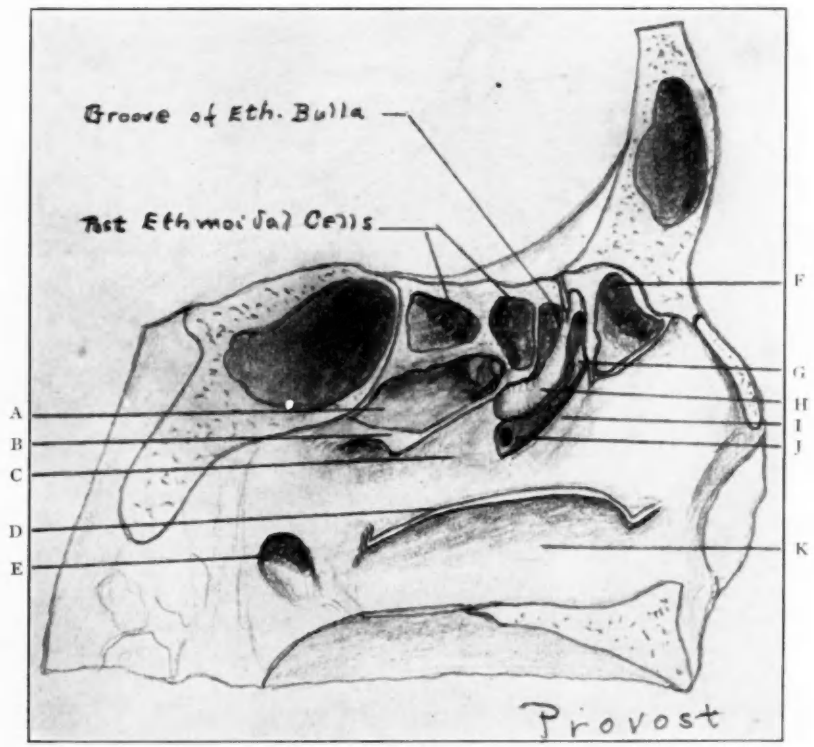


FIG. 15.—Student drawing (Provost) from a wet specimen.

A. Superior meatus. B. Attachment middle turbinate. C. Middle meatus. D. Cut inf. turbinate. E. Eustachin orifice. F. Agger nasi cell communicating with unciform groove. G. Unciform groove where it goes into the agger nasi cell. H. Bulla. I. Unciform process. J. Accessory ostium (antrum). K. Inf. meatus.

corresponding choana. Excessive secretions from either the anterior or the posterior ethmoidal cells disregard purely anatomical channels, fill the whole nasal cavity and appear both at the vestibule of the nose and in the pharynx.

The Blood Supply of the Labyrinth

The chief blood supply of the labyrinth enters the back door and the door is the spheno-palatine foramen placed in the upper part of the palate bone and bounded above by the body of the sphenoid. Through this is a branch of the internal maxillary artery traversing the zygomatic fossa and the terminal portion of this fossa, the pterygoid palatine fossa, reaches the nasal cavity to divide into two branches, one for the outer wall of the

FIG. 16.—The upper drawing shows an ethmoidal cell mounding into the frontal sinus (frontal bulla.)

The lower drawing shows an ethmoidal cell from the top of the ethmoidal bulla running forward to the orbital run and making an accessory frontal sinus.

nose which includes the ethmoidal labyrinth, the other which crosses the front face of the sphenoid, to supply the septum and on its way there, the sphenoidal sinus. The spheno-palatine artery is the chief blood supply of the nose. Anteriorly through the ethmoidal foramena, branches of the ophthalmic artery by means of the posterior and the anterior ethmoidal foramena, reach the labyrinth.

The veins connect liberally through the cribriform lachrymal bone with the plexus of veins about the lachrymal sac. They connect with the main orbital veins and so with the cavernous sinus. There is a connection posteriorly with the pterygoid plexus of veins and so again with the cavernous sinus. Posteriorly also the veins connect with the palatine veins. Through

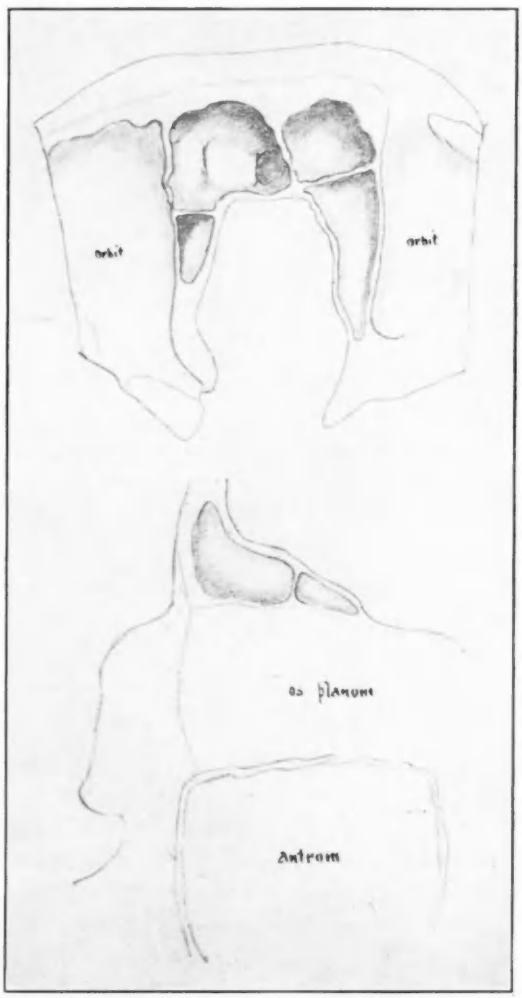


FIG. 17.—Student drawing (Wright) from a wet specimen to show a fronto ethmoidal cell in series with the frontal sinus.

the off-shoots of the cavernous sinus and the connection across the bridge of the nose between the angular veins the venous circulation of both orbits are closely associated posteriorly and anteriorly and the ethmoidal veins connect with the orbital veins.

The Nerve Supply of the Labyrinth

The chief nerve supply of the labyrinth also gains its entrance from behind and again through the spheno-palatine foramen. The chief nerve supply comes from Meckel's ganglion placed in the pterygo-palatine fossa, a short distance to the outside of the spheno palatine foramen. Anteriorly through the anterior palatine foramen the naso-cilliary, a branch of the ophthalmic gives a supply to the labyrinth.

On the inner surface of the ethmoidal labyrinth in grooves leading to corresponding perforations in the cribriform plate lie the outer branches of the olfactory nerve, the nerve of smell.

The anterior ethmoidal cells which are of surgical importance are first, the agger nasi. This is present in a third of specimens and is found in the extreme anterior superior end of the middle turbinate. Its inner boundary is the flaring upper end of the unciform process. Its outer boundary is the inner surface of the ascending process of the superior maxilla at the point where the ridge called the agger nasi occurs. In series with the agger nasi cell and posterior to it is the lachrymal cell. This is fairly constant. Its outer wall is the lachrymal bone. It has to be entered in the intra nasal or the orbital operation on the tear sac. The frontal bulla is another anterior ethmoidal cell of surgical importance. It is a high cell which mounds into the floor of the frontal sinus and is always in relation with some part of the upper opening of the naso-frontal duct. When it is present it makes it easier to enlarge the opening of the naso-frontal duct working either from the nose or from the frontal sinus. At times this cell reaches the orbital rim to the outside of the frontal sinus and makes an accessory frontal sinus. *(Figs. 16-17.)*

The ethmoidal bulla may consist of one ethmoidal cell, but generally contains two. The bulla is the most accessible part of the labyrinth anteriorly and inferiorly. A large bulla can block the lower half of the unciform groove and hinder drainage from the ostium of the antrum. Medially the bulla is covered by the inferior overhang of the middle turbinate. The upper cell of the bulla may grow forward to the rim of the orbit and make an

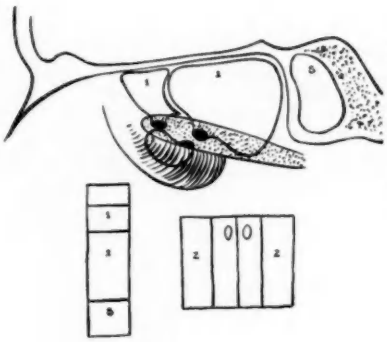
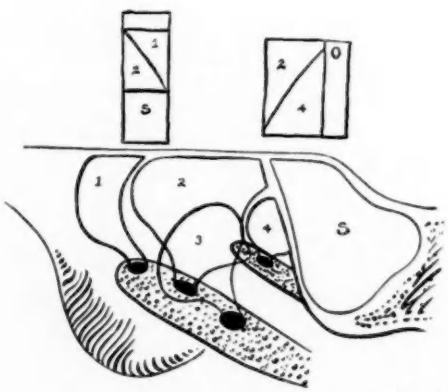


FIG. 18.—Drawing from a specimen in which two posterior cells make up the major part of the posterior half of the ethmoidal labyrinth. Very little curetting would be necessary to turn the posterior half of the ethmoidal labyrinth into a single cavity.

The first small drawing below shows the area of the roof of the ethmoidal labyrinth occupied by the summits of cells 1 and 2. The second small drawing shows the area of the front face of the sphenoidal sinus, which has a common wall with the posterior ethmoidal cell.



—Sieur and Jacob

FIG. 19.—Drawing from a specimen in which the third and fourth meatus are present. Three ethmoidal cells open into the third meatus, and one into the fourth. The size and the arrangement of the posterior cells is well shown: Cell No. 1 runs outward to the os planum, has its base there and then runs to the roof of the ethmoidal labyrinth. Cell No. 2 is larger, runs outward to the os planum, and the upward to the roof of the labyrinth. It also runs backward and has a common wall with the upper outer half of the front face of the sphenoidal sinus. Cell No. 3 runs outward to the os planum, but does not reach the roof of the labyrinth. Cell No. 4 has a common wall posteriorly with the inner lower half of the front face of the sphenoidal sinus.

The small figure above on the right shows the relationship of cells 1 and 2 with the roof of the ethmoidal labyrinth. The small figure on the left shows the relationship of cells 2 and 4 with the front face of the sphenoidal sinus.

accessory frontal sinus. A more important extension of this cell is upward and outward over the orbit, behind the frontal sinus, and in series with it. Such a cell is called a fronto-ethmoidal cell. When undiscovered and not drained it is one of the most frequent causes of the failure of the radical frontal sinus operation. An X-ray should always be taken in the Water's position in order to show whether or not it is present.

It is not uncommon to have two large cells make the whole posterior part of the labyrinth. Three or four cells are more usual. The most posterior cell is the important one. When the third meatus is the highest meatus this cell springs from the inferior portion of the third meatus and its posterior wall is a common wall with the outer half of the front face of the corresponding sphenoidal sinus. This cell is near the optic nerve—one to five mm. It is common to have the optic nerve mound into the superior outer angle of this cell. The wall of the mound may be paper thin. *(Figs. 18-19-21.)*

In 62 per cent of cases there is a fourth meatus and in 75 per cent of the cases in which a fourth meatus is present a cell arises from it. This cell generally takes the upper part of the common wall of the front face of the sphenoid, and the cell from the lower part of the third meatus the remaining part. *(Fig. 20.)*

The most posterior ethmoidal cell frequently is prolonged backward in an extension over the sphenoidal sinus. It may be prolonged backward on the outer wall of the sphenoidal sinus. Such a prolongation of the posterior ethmoidal cell may be so large as to be the surgical sphenoidal sinus of that side, the anatomical sinus being a small affair. This cell is always liable to confuse the operator unless the X-ray, alone or aided by the installation of lipiodal, picks it out. In cadaver operating this cell is often found unopened when the head is sectioned antro-posteriorly and the operated field examined, and this in spite of repeated warnings on the part of the instructor not to miss it. One is especially liable to miss it, I feel, by the approach to the ethmoid through the antrum.

The posterior ethmoidal cell may mound down into the antrum and even reach the floor of the sinus, making what is called a double antrum. I have found but one specimen of this. *(Fig. 22.)*

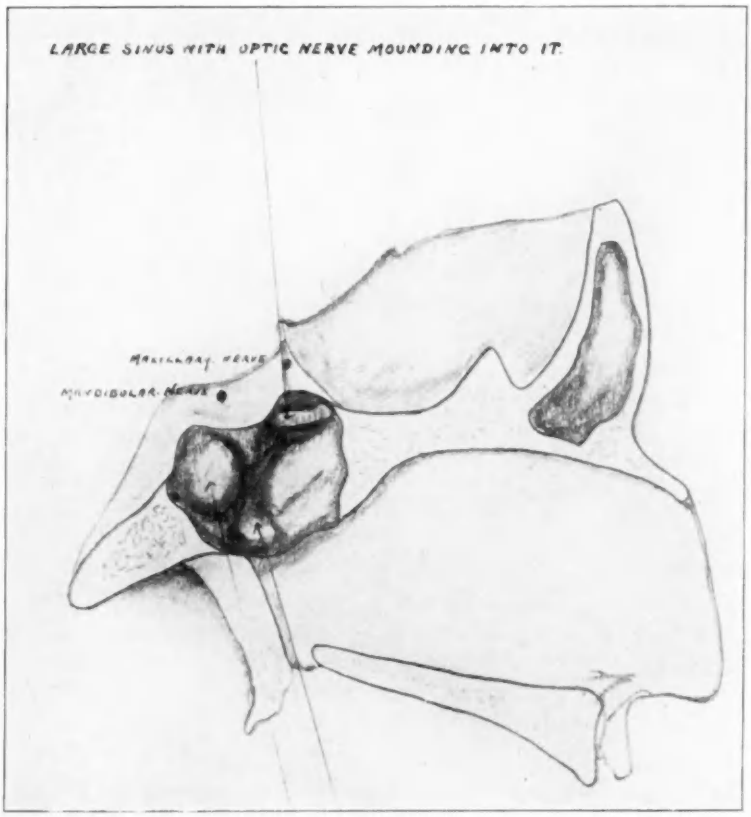


FIG. 20.—Student drawing (Wright) to show a large Sphenoidal Sinus with the Optic Nerve mounding into it. There are prolongations extending into the great wing and into the pterygoid process.

Surgical Approaches to the Ethmoidal Labyrinth

The surest method of reaching the naso-frontal duct is to make a curette plunge through the agger nasi cell and then curette forward until the posterior surface of the ascending process of the superior maxilla stops the advance of the curette. *(Figs. 23-4-5-6-7.)* A probe passed into such an opening and carried up along the posterior surface of the ascending process of the superior maxilla finds the naso-frontal duct in a great majority of cases and enters the frontal sinus. *(Figs. 7-8-9.)* If there is no agger nasi cell the plunge should be made through the extreme upper anterior end of the middle turbinate. A common mistake in executing this procedure is not to go high enough on the middle turbinate, or far enough back to miss the inner surface of the ascending process. The curette plunge is the writer's favorite method of beginning the intranasal removal of the anterior ethmoid cells. If the curette plunge into the anterior part of the middle turbinate followed by the introduction of the probe does not result in finding the naso-frontal duct the curette is reintroduced and brought forward and the posterior surface of the ascending process of the superior maxilla is curetted free from cells. Another try is then made with the probe. If it fails to pass the curette is reintroduced facing backward and all of the anterior cells are curetted. In a saucer shaped frontal sinus the beginning of the naso-frontal duct is often placed far back. With this in mind the operator, after finishing curetting the anterior ethmoidal cells, introduces the probe to the posterior limit of the operated cavity, carries the point to the roof of the cavity posteriorily and then brings it forward along the outer edge. As has just been said, however, the curette plunge followed by curetting forward to the ascending process is successful in finding the naso-frontal duct in fully 90 per cent of cases. This little procedure which the writer introduced many years ago proved to be a most useful contribution to the applied anatomy of the ethmoidal labyrinth.

The Orbital Approach to the Anterior Ethmoidal Cells

Killian taught us to remove a section of the ascending process of the superior maxilla in front of the lachrymal bone, in order to reach the anterior cells from the front. Many years ago the writer gave up this procedure and entered the laby-

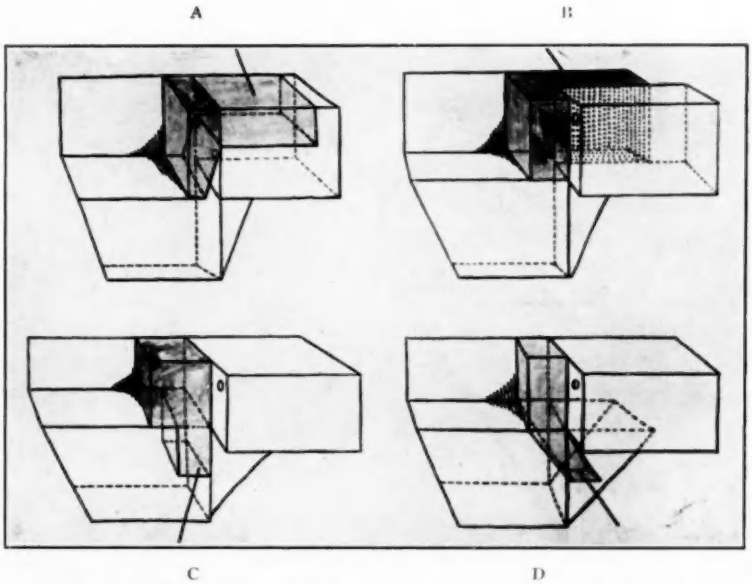


FIG. 21.—Diagrammatic drawing to show the anteriors of the most posterior ethmoidal cell. A, extension backward over the sphenoidal sinus; B, extension backward and lateral to the sphenoidal sinus; C, extension downward into antrum; D, extension into the ptregoid process.

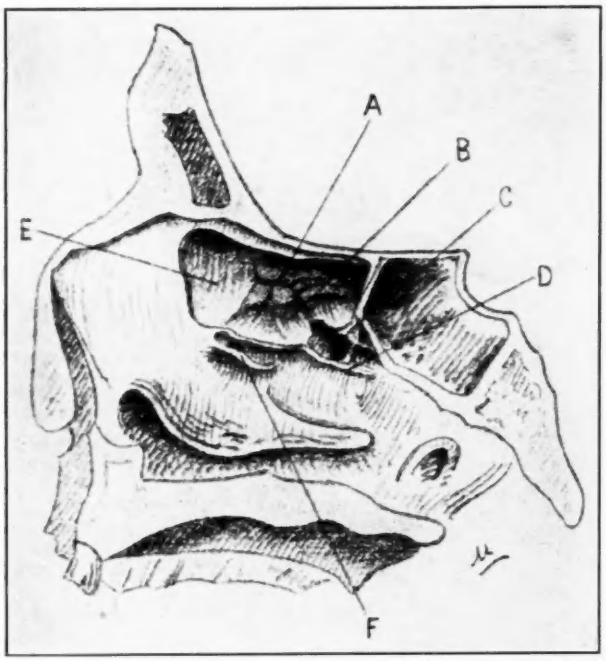
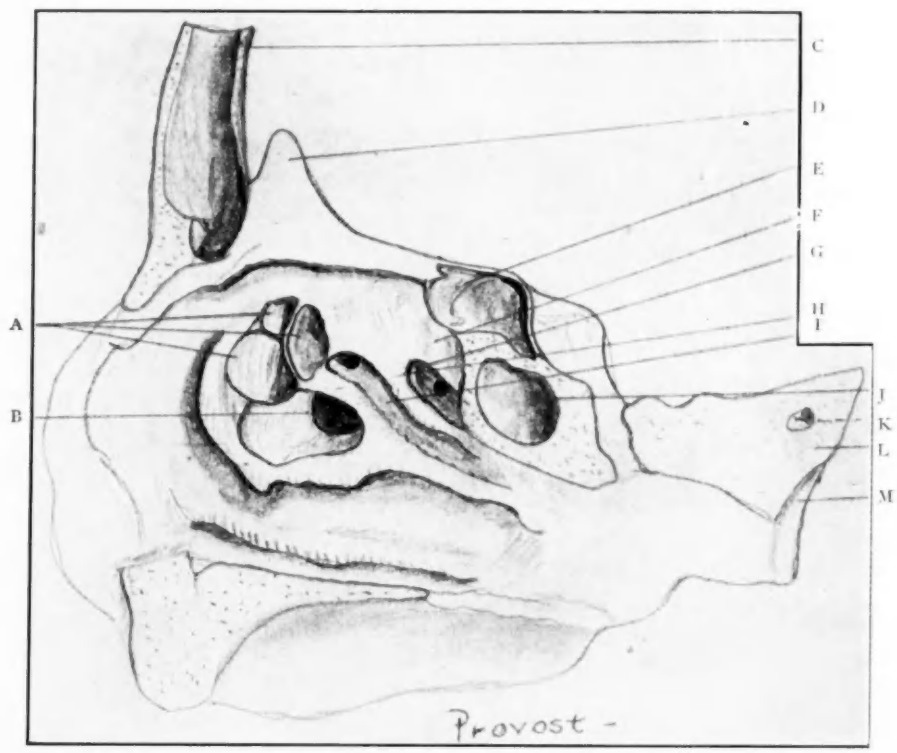


FIG. 22.—In this specimen the ethmoidal labyrinth overhangs the antrum markedly and one posterior cell projects downward into it. A. os planum; B. Antral Overhang; C. post-ethmoidal cell overhanging the antrum; D. posterior part of the attachment of the middle turbinate; E. anterior portion of ethmoidal labyrinth; F. unciform process.



A. Anterior ethm. cells. B. Cell within middle turbinate ostium into middle meatus opposite infundibulum. C. Frontal sinus. D. Crista galli. E. Posterior ethmoidal cell. F. Supreme turbinate. G. Supreme meatus ostium into a post ethm. cell. H. Superior turbinate. I. Superior meatus ostium into a post. ethm. cell. J. Sphenoidal sinus. K. Cochlea. L. Part of temporal bone. M. Region of jugular bulb.

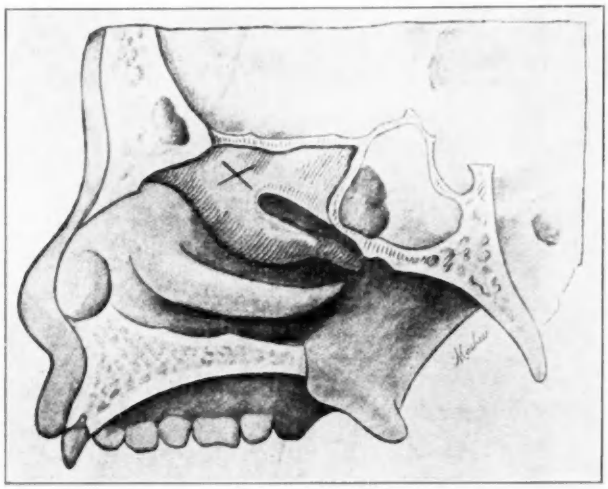


FIG. 24.—Drawing showing the middle and superior turbinates. The cross indicates the point at which the curette is carried into the ethmoidal labyrinth. The portion of the middle turbinate below the summit of the third meatus is called in this paper the lower overhang, and the portion above and in front of this point, the upper overhang. The cross is placed on the upper overhang.

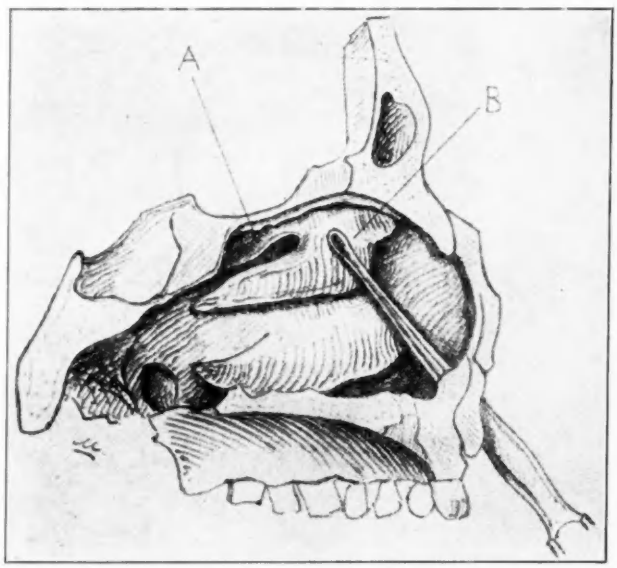
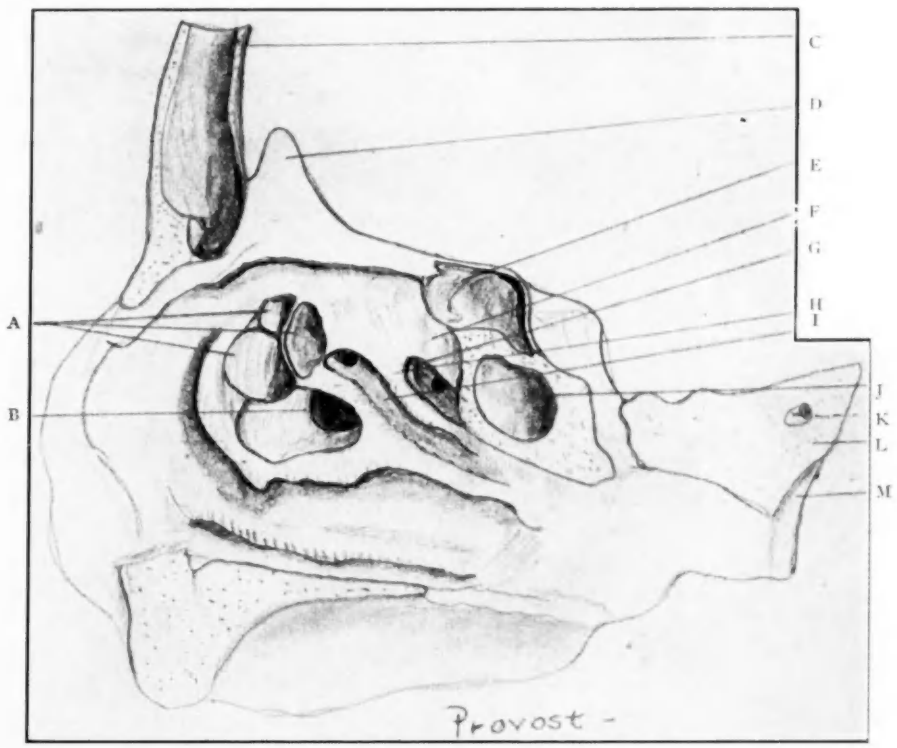


FIG. 25.—The writer now begins the ethmoidal operation by removing the inferior overhang of the middle turbinate with scissors and then piercing the superior overhang as in the cut. There are less tags to remove if this is done. The curette is placed against the upper overhang of the middle turbinate for the initial plunge into the labyrinth. A, superior turbinate; B, middle turbinate. If the curette does not enter readily it should be pushed a little higher up and a bit further back.



A. Anterior ethm. cells. B. Cell within middle turbinate ostium into middle meatus opposite infundibulum. C. Frontal sinus. D. Crista galli. E. Posterior ethmoidal cell. F. Supreme turbinate. G. Supreme meatus ostium into a post ethm. cell. H. Superior turbinate. I. Superior meatus ostium into a post. ethm. cell. J. Sphenoidal sinus. K. Cochlea. L. Part of temporal bone. M. Region of jugular bulb.

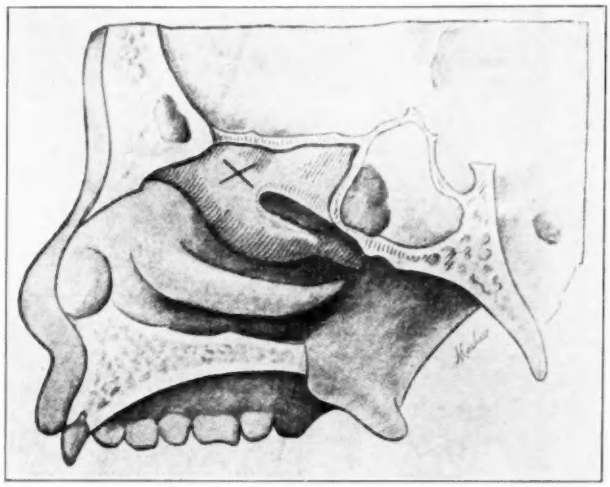


FIG. 24.—Drawing showing the middle and superior turbinates. The cross indicates the point at which the curette is carried into the ethmoidal labyrinth. The portion of the middle turbinate below the summit of the third meatus is called in this paper the lower overhang, and the portion above and in front of this point, the upper overhang. The cross is placed on the upper overhang.

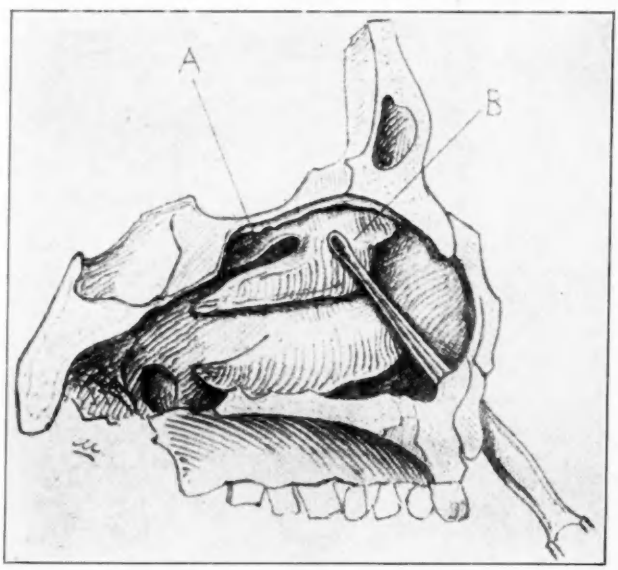


FIG. 25.—The writer now begins the ethmoidal operation by removing the inferior overhang of the middle turbinate with scissors and then piercing the superior overhang as in the cut. There are less tags to remove if this is done. The curette is placed against the upper overhang of the middle turbinate for the initial plunge into the labyrinth. A, superior turbinate; B, middle turbinate. If the curette does not enter readily it should be pushed a little higher up and a bit further back.

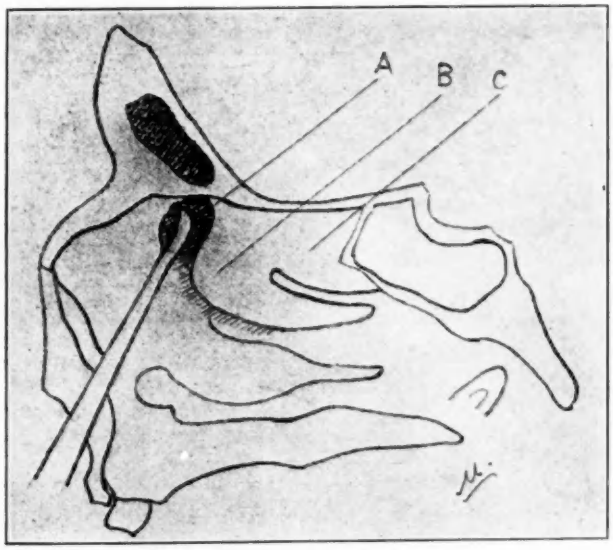


FIG. 26.—Position of the curette (face turned forward) in the act of curetting the posterior surface of the ascending process of the superior maxilla. A, ethmoidal labyrinth; B, middle turbinate; C, superior turbinate.

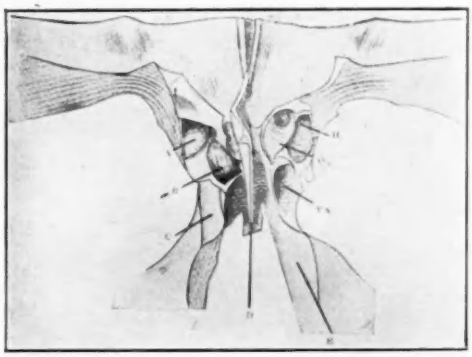


FIG. 27.—Drawing to show the relationship of the naso-frontal duct to pockets from the frontal sinus and to ethmoidal cells developed in the ascending process of the superior maxilla.

On the left there is a good sized pocket or prolongation. This springs from the frontal sinus and is below and internal to the duct. There may be a regular cell developed at this place. It is not infrequent to find two such cells. When a prolongation or a cell is present, if the examining probe is carried upward and inward, its point catches in the prolongation. If, however, the point of the probe is directed outwards, the opening of the naso-frontal duct is entered.

On the right the specimen shows two ethmoid cells, the upper developed in the frontal bone and the lower one in the ascending process of the superior maxilla.

A, Naso-frontal duct. B, Pocket of frontal sinus; C, Lacrimal groove; D, Septum; E, Ascending process of superior maxilla; F, Ethmoidal cell; G, Ethmoidal cell; H, Naso-frontal duct.

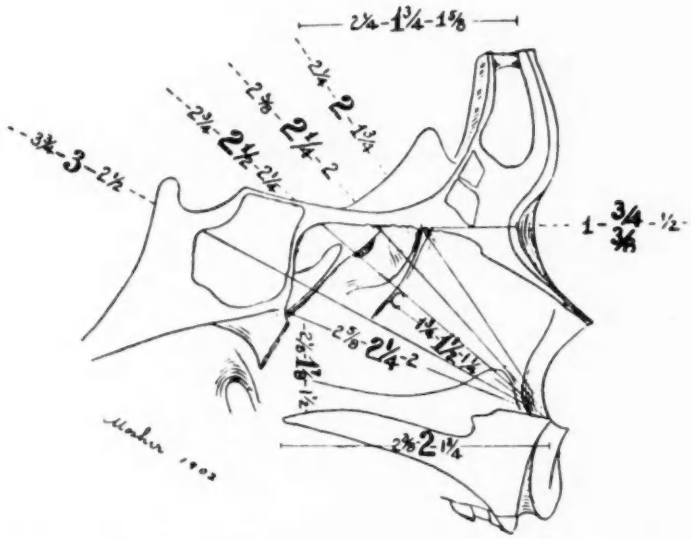


FIG. 28.—Operating distances obtained from the measurement of sixty-four half heads. The lachrymal duct is not given on this diagram. The average distance is one inch.

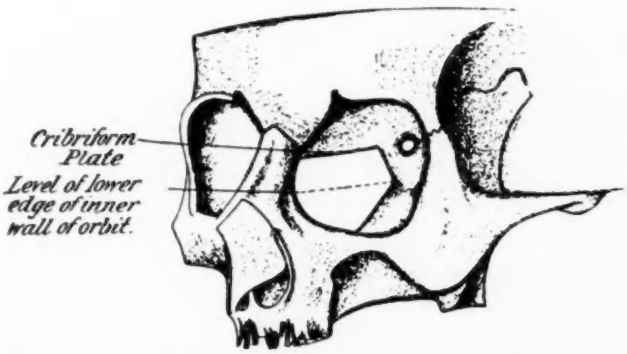


FIG. 29.—Drawing showing the level of the cribriform plate. The inner wall of the orbit and a part of the floor have been taken away.

rinth through the lachrymal bone. This is thin and, of course, easily perforated. Once through the lachrymal bone the operator can work forward and remove as much of the ascending process as he pleases. This saves some ten minutes of time, is simple and more direct.

It was formerly the custom to remove the os planum only as far back as the anterior ethmoidal foramen, that is, to the limit of the anterior cells. The further the operator goes back, however, the easier it is to expose the posterior ethmoidal cells and to make out their extensions. Lynch has called attention to the advantage of the extensive removal of the os planum. Of course the removal should not be carried as far as the optic foramen.

The Removal of the Floor of the Frontal Sinus

In the early days of the radical operation on the frontal sinus, it was customary to remove the floor of the sinus by chiseling from within the sinus downward into the orbit. A surer method of removing the whole of the floor of the sinus is to work within the orbit from below upward and outward. Having gained access to the ethmoidal cells through the lachrymal bone and located the naso-frontal duct, a moderate amount of the os planum is removed. This exposes the inner part of the floor of the fontal sinus where it makes the internal angular process of the frontal bone and articulates with the lachrymal bone and the os planum. The author has found it a very useful surgical conception to consider the floor of the frontal sinus as the upward and outward extension of the os planum. Therefore, after a sufficient amount of the os planum has been removed to allow the probe to ascertain the extent of the floor of the sinus, the operator works upward and outward and backward until the whole of the floor has been taken away. This conception of the floor of the frontal sinus as the upward and outward prolongation of the os planum is simple enough, but the writer was many years in thinking of it. It has proved of great help, however, in operating.

If a fronto-ethmoidal cell is present, the floor of this also is made by the upward outward extension of the os planum, and the floor of the cell is removed by working upward and outward after the manner employed in the removal of the floor of the frontal.

FIG. 30.—Drawing to show the horizontal planes of the cribriform plate, the ostium maxillare, the lachrymal duct, and the Eustachian tube.

FIG. 31.—Cast of ideal septum to show measurements.

At least twenty-five years ago Jansen was accustomed to operate on the ethmoidal labyrinth by way of the canine fossa of the antrum and the membranous area of the base of the antrum. He also approached the sphenoid in the same way. In these days of local anæsthesia and adrenaline the procedure is much easier to carry out because it is less bloody than in the old days. At least one writer has lately advised the revival of the procedure. I regularly use this procedure in a joint operation on the ethmoid and the antrum, checking up the position of my instrument introduced through the orbital opening into the ethmoid by inspection through the antrum.

Of late years operators on the ethmoidal labyrinth have in increasing numbers successfully employed local instead of general anæsthesia. This method of operating has increased the importance of the anatomical location of the nerves and arteries which supply the labyrinth. Fortunately both are very definitely placed. First, the anterior ethmoidal foramen carries from the orbit to the ethmoidal labyrinth the anterior ethmoidal artery and the naso-cilliary nerve. The posterior ethmoidal foramen carries the posterior ethmoidal artery. Through the spheno-palatine foramen from Meckel's ganglion and from the internal maxillary artery come the nerve and blood supply of the labyrinth posteriorly.

Under general anesthesia operators have generally disregarded the ethmoidal arteries. Sewall, however, has brought home to the profession the great advantage of tying the anterior and posterior ethmoidal arteries and in operating on the sphenoid the advantage of tying the spheno-palatine.

Nasal Measurements

A knowledge of fundamental nasal measurements is a great comfort to the operator, especially to the beginner. To make them of the greatest value, each student of nasal anatomy should make at least one set of measurements for himself on a wet specimen. *(Figs. 28-29-30.)*

Post graduate students seize with avidity upon a chart of nasal measurements which the writer got out for his own guidance at the beginning of his teaching many years ago. This chart and one or two others of nasal measurements are included in this paper for reference.

In what might be called the ideal adult septum it will be found in measuring from the base of the anterior nasal spine (see Fig. 31) that the measurement of 2 in. is the measurement to certain very vital and definite points. In other words, the operator should never go beyond 2 in., or 5 cm., in the nose without being on his guard. For instance, the length of the base of the septum in 2 in. The same measurement carries the probe to the middle of the front face of the sphenoid and at the mid point of the ethmoidal labyrinth, to the cribriform plate. The

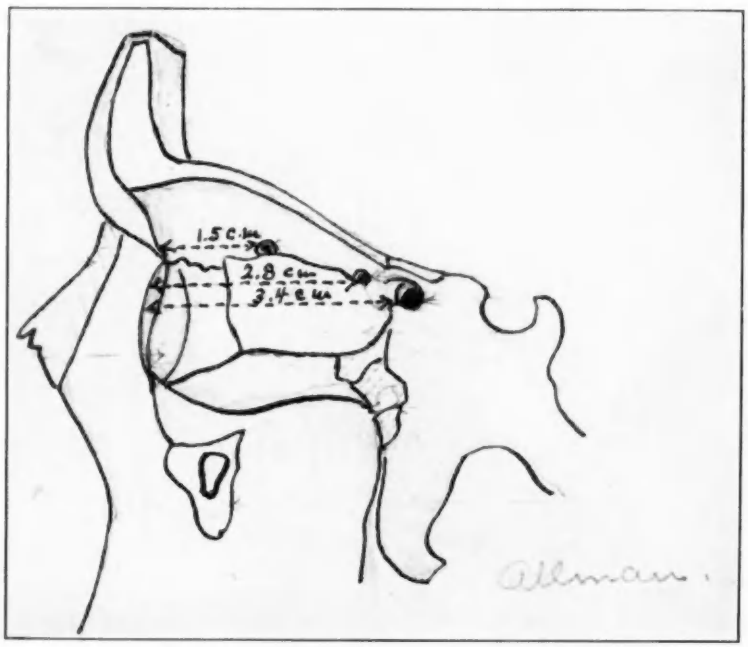


FIG. 32.—Student drawing (Allman) from wet specimen to show distances on the inner wall of the orbit.

vertical measurement of the nasal cavity, as will be seen by reference to Fig. 31, is slightly less than 2 in.

By way of review I will end this short paper by giving the quiz questions and their answers which are used in my post graduate class work. The quiz is followed by a tabulation of the findings in 20 half heads, the number on which a recent class, as part of their routine work, dissected the ethmoidal labyrinth.

Quiz on the Ethmoid

How much of the lateral wall of the nose is made by the ethmoidal labyrinth?

The upper half. The lower half is made by the nasal wall or the base of the antrum.

How much fluid will the ethmoidal labyrinth hold?

8-10 cc—3 drachms.

What is the shape of the labyrinth?

Pyramidal—wider behind than in front—wider above than below.

What is the width of the labyrinth anteriorly?

Half a cm. to a cm.

What is the width posteriorly?

1½cm.

What is the antero posterior length of the labyrinth?

3 to 4 cm.

What is the height of the labyrinth?

2—2½cm.

What part of the labyrinth is most accessible from the nose?

The agger nasi cell if present—otherwise the ethmoidal bulla.

Does the cribriform plate make the roof of the ethmoidal labyrinth?

No, a prolongation of the orbital plate of the frontal makes the roof of the ethmoidal labyrinth.

What does the cribriform plate make the roof of?

The olfactory slit.

The ethmoidal cells are present at the 7th month of fetal life. There is no uniform development. One ethmoidal labyrinth may be larger than the other. The cells empty into the meatus from which they spring. The cells are bottle shaped. The bases of the cells are on the os planum and the lachrymal bone. Many cells reach the base of the skull. The cells vary from 3 to 15.

Operations have been done on the labyrinth as early as three years. (Meyer).

What anatomical structure divides the anterior from the posterior ethmoidal cells?

The basal lamella or the attachment of the middle turbinate. The anterior ethmoidal foramen is at the upper end of the attachment of the basal lamella of the middle turbinate.

What forms the outer or the orbital wall of the anterior ethmoidal cells?

The lachrymal bone. Therefore in approaching the anterior ethmoidal cells from the orbit you approach them through the lachrymal bone.

What forms the outer or the orbital wall of the posterior ethmoidal cells?

The os planum.

What forms the inner or nasal wall of the anterior half of the ethmoidal labyrinth?

The inner walls of the anterior ethmoidal cells. That is, the agger nasi cell—the lachrymal cell—the infundibular cells—the cells of the ethmoidal bulla. All of these cells are overlain by the upper anterior part of the middle turbinate.

What forms the inner wall of the posterior ethmoidal cells?

The superior turbinate—the third and fourth turbinates.

What is the posterior limit of the labyrinth?

The outer half of the front face of the corresponding sphenoidal sinus.

What is the upper anterior limit of the labyrinth?

The posterior surface of the ascending process of the superior maxilla.

What is the lower anterior limit of the labyrinth?

The ethmoidal bulla.

What are the most important relationships of the anterior ethmoidal cells?

With the lachrymal bone—the floor of the frontal sinus—the hiatus semilunaris or unciform groove and through this with the nasofrontal duct and the ostium of the antrum.

What are the most important relationships of the posterior ethmoidal cells?

With the outer half of the front wall of the corresponding sphenoidal sinus—with the posterior half of the inner wall of the orbit—with the optic nerve.

What is the thickness of bone between the optic nerve and the most posterior of the ethmoidal cells?

Half a mm to 5 mm. The nerve may mound into the cell.

What are the most common extensions of the anterior ethmoidal cells?

Anteriorly to the inside of the ascending process of the superior maxilla making the agger nasi cell—upward into the frontal sinus, making a frontal bulla—the extension upward and outward of the superior cell of the ethmoidal bulla making a cell in series with the frontal sinus—the fronto-ethmoidal cell. The ethmoidal cells are continually getting out of bounds.

What are the most common extensions of the posterior ethmoidal cells?

Backward over the sphenoidal sinus—backward to the side of the sphenoidal sinus. Rarely downward into the antrum making the so called double antrum. Into the middle turbinate making a cystic turbinate.

Dehiscences of the walls of the labyrinth.

The books state that dehiscences are not infrequent in the os planum. The lachrymal bone is normally cribriform with small dehiscences.

The Blood Supply—

The ophthalmic artery from the internal carotid. The anterior and posterior ethmoidal arteries. The spheno-palatine artery from the internal maxillary.

Nerve Supply—

Meckel's ganglion—the nasal branch of the ophthalmic.

Veins—

Connect with the facial—the inferior ophthalmic and cavernous sinus—the pterygoid plexus and so with the cavernous sinus—with the pharyngeal veins.

Lymphatics—

Connect with the lymphatics of the cranial cavity especially by way of the sheaths of the olfactory nerves. They drain chiefly into the gland in the pharyngo-maxillary fossa which is placed on the body of the second vertebra—the gland which suppurates in retropharyngeal abscess.

The Naso-frontal duct—

When is a naso-frontal duct not a duct?

Usually there is no duct. In most cases the unciform groove ends blindly or in an ethmoidal cell.

What is the best method of reaching the opening of the duct into the frontal sinus?

The approach through the agger nasi cell or, if this is not present, through the upper anterior part of the middle turbinate.

Number of half heads dissected	20
Number of agger nasi cells found	8
Unciform groove continuous with the naso-frontal duct	3
Unciform groove ending blindly	17
Fronto-ethmoidal cell	1
Greatest number of anterior ethmoidal cells	5
Smallest number of anterior ethmoidal cells	2
Greatest number of posterior cells	3
Smallest number not recorded.	
4th Meatus present in	5
Longest labyrinth	4 cm.
Shortest labyrinth	2.75 cm.
Posterior ethmoidal cell running back over the sphenoid	1
Posterior ethmoidal cell running backward and lateral to the sphenoid	4

SYMPOSIUM ON THE ETHMOID.

PATHOLOGY AND DIAGNOSIS

ROSS HALL SKILLERN, M. D.
PHILADELPHIA, PA.

Ethmoiditis, both the hyperplastic and suppurative varieties have received so much publicity that to occupy your time with descriptions of the gross pathology of these lesions would be as discourteous as it would be superfluous. We all, of course, know and recognize that the hyperplastic variety may occur without a vestige of discernable suppuration, and the suppurative variety continue for years without the appearance of a polyp, but that these two conditions can and do occur simultaneously, no one who has had experience which is worthy of the name can deny. That suppuration may continue for a period of years without the formation of hyperplastic tissue is now so well established that it has become one of the postulates of sinuology. As a matter of fact, suppuration tends to atrophic changes rather than the hypertrophic variety.

There is, however, one point in the gross pathology of the ethmoid that I have found of unfailing significance, and that is, the general appearance of the middle turbinate. When this body is healthy, it seems to fit in nicely with the underlying structures, the mucosa is smooth and taut, of a pinkish grey, dry, more or less shiny, does not bleed easily, with no traces of any sort of a secretion. Now in my experience, as soon as the ethmoid becomes infected, the middle turbinate begins to reflect the condition almost in direct ratio to the severity of the infection. These changes in the beginning are very slight and at first blush are usually overlooked, unless one especially has them in mind, and even then, it often requires the closest scrutiny and frequent comparison with the other side before one is convinced that something is amiss.

These aberrations from normal concern first, the position of the turbinate, second, the size of the turbinate and third, the condition of the turbinate mucosa. The position and size of the

turbinate are only influenced after the infection has secured a firm hold, therefore rather late in the disease. The changes in the turbinal mucosa, however, occur comparatively early, which fact alone makes their recognition of vital importance. Ordinarily on anterior rhinoscopy, the middle turbinate cannot be thoroughly inspected, due to the close proximity of the neighboring structures. There is one portion, however, that can always be brought into view, and that is the lower border in its entire length. Fortunately, this is the very portion that shows these significant changes when infection takes hold, the deviation from the normal being in proportion to the severity of the inflammation.

The usual early change is one of color. The mucosa along the turbinal edge seems to gradually blanch until it assumes a translucent appearance, not unlike a beginning polyposis. This may be so gradual as to give the impression that the process has become stationary, but such is never the case, as the changes are slowly but surely creeping upward along both sides of the turbinate. The longer this continues, the greater the metamorphosis, until finally true polypoid changes are present. Different infections seem to present different pictures. One of the most striking of these is a peculiar mottled appearance on this lower edge and is significant, to my mind, of a deep seated and rather serious infection. In many cases with marked evidences of toxic absorption of particular gravity (in which the heart or kidneys are involved) this appearance of the lower border of the turbinate is present. The body of the ethmoid itself may show little macroscopic change from normal in conjunction with these turbinal changes, however, I have noted that when opened, the bleeding is much freer than normal (showing excessive congestion) and a culture by platinum look invariably shows a pure culture of a staphlococcus.

Now a word regarding the microscopic pathology. Here we are first concerned with the differentiation between the hyperplastic and suppurative varieties. When these types are pure, recognition is not difficult as they show very dissimilar changes as follows:

Hyperplastic	Suppurative
Ciliated epithelium remains unchanged except where friction exists as between two polyps.	Epithelium looses cilia early.
Connective tissue meshes dilated.	Connective tissue shows fibrous formation consequently retracted.
Glands hypertrophied (primarily.)	Glands atrophied.
Reabsorptive changes in bone (osteoporosis.)	New bone formation not infrequent.

When a purely hyperplastic variety becomes infected with formation of pus, then of course the picture presented shows characteristics of both, provided the suppurative process has continued for a sufficient length of time, however, when this occurred the diagnosis has long since ceased to be in doubt. Slides 1 to 8.

Diagnosis

From what has been said it will at once be seen that a fairly intimate knowledge of the pathology is essential before one can expect to recognize minute changes sufficient to deduce a reliable diagnosis in the earlier stages of ethmoid infection. The old postulate that in order to arrive at a reliable diagnosis we must find pus and follow it to its source is as obsolete as the dodo, for when pus has formed we know that the general system has reacted against the infecting organisms and is probably holding its own. The recognition and diagnosis of this condition would be plain to the merest tyro; what we should do is to recognize the infection before it goes on to the purulent stage thereby placing ourselves in position to prevent either its spread to neighboring cells or permanent pathologic changes taking place in the ethmoidal structures. Now how can this be done? Partly by studying the changes in the position, shape and mucosa of the middle turbinate and keeping in mind not only those conditions that occur in text books, but those that we continually meet with in our practices. Let us then consider the text book conditions with those that we will call actualities in want of a better term.

TEXT BOOK CONDITIONS

a. Empyema of an anterior cell or cells.
b. Empyema of a posterior cell or cells.
c. Combined empyema.
d. Hyperplasia.
e. Combined hyperplasia with suppuration.
Slides 9—

ACTUALITIES

a. Generalized infection of mucosa which may occur in a small or large area; rarely empyema of one cell or sharply defined group of cells.
b. General purulent infection resembling atrophic rhinitis.
c. Hyperplasia in various stages.
d. Combined hyperplasia and suppuration.

a. Empyema of Anterior Cell or Cells.

In my experience, despite our previous teachings, this is one of the rarest occurrences per se that we meet within nasal affections. When a purulent frontal sinusitis exists, of course, an extention downward of the inflammation is all that is necessary to infect the mucosa of any cells emptying into the infundibulum. This combination as an actuality is, however, more apparent than real for who has ever seen the persistence of suppuration in an infundibular cell after the frontal sinusitis has abated? The one exception to this rule appears to be the ethmoidal bulla, for very occasionally one may find it necessary to thoroughly open this cell to evacuate the pent up pus.

b. Empyema of Posterior Cell or Cells.

This form is somewhat commoner than the preceding but as actual localized abscess is by far the exception.

c. Combined Empyema.

Excepting the form which will be described later, this is one of the rarest affections that the ethmoid shows.

d. Hyperplasia.

The commonest of ethmoid affections. To enter into a detailed description of polyps and polypoid hypertrophis at this time would be as tiresome as it would be unnecessary. All of us have studied these conditions both clinically and in the privacy of our studies, so that anything I might bring out here would only be in the nature of a repetition. Let us however thoroughly recognize that in contra-distinction to the combined empyema, the hyperplastic form represents unquestionably the commonest

variety of ethmoidal disease that we are called upon to treat. Furthermore, despite the views of many authorities, I simply cannot believe that this form of affection is, at least primarily, due to micro-organismal activity.

c. Combined Hyperplasia with Suppuration.

This form represents an old and very persistent process being the final stage of a chronic hyperplasia in which purulent infection had occurred which in turn had also become chronic. Nowadays we only see these cases in patients who have suffered with hyperplastic ethmoiditis for years and have been operated and reoperated upon many times. As we have now briefly covered the conditions as laid down in text books, let us now consider those that we encounter more frequently in our offices.

a. Generalized infection of mucosa which may occur in small or large area; rarely empyema of one cell or sharply defined group of cells.

Despite the fact that this form of disease is seldom noted, nevertheless, I believe it to be the commonest type of ethmoidal infection capable of causing serious systemic disturbances. In the beginning, it is usually so inconspicuous as to be entirely overlooked and indeed may even be present inside the capsule without disclosing itself by any outward manifestation. As a rule, however, the appearance of the ethmoid base or the middle turbinate will give one a clue as has been noted under the pathology. That this condition can and may go on to form a collection of pus in one or more cells is perfectly possible and in the earlier days was apparently of ordinary occurrence, but since the sinuses have become common property with the laity, the infection is no longer permitted to progress to this stage, but is usually taken in hand at the onset of the primary symptoms with an anticipated diagnosis of "something the matter with my sinuses."

b. General Purulent Infection Resembling Anthrophic Rhinitis.

The older writers were obsessed with the idea that atrophic rhinitis (ozena) under certain circumstances caused purulent Sinusitis, and indeed Hajek devoted an entire chapter in his book to this subject. I, of course, accepted this a dictum ex cathedra until, from the repeated dissimilarity of symptoms, it gradually began to occur to me that these cases were different from the usual atrophic cases seen in profusion at that time in our clinics. In the first place, the typical odor was always absent and, secondly, the atrophy seemed to be limited to the inferior turbinals,

the ethmoid region being swollen and hypertrophied. I am now convinced and have been for a long time that this affection represents an aberrant type of purulent ethmoiditis which though comparatively rare, seems to have been noted by everyone who has been interested for any time in sinuology.

c. Hyperplasia in various stages.

d. Combined hyperplasia and Suppuration.

This form represents the result when infection occurs and finds a permanent lodging in an advanced case of hyperplastic variety. This is caused by the polyps and hypertrophied tissue becoming so wide spread as to occlude the upper and middle nasal passages then infection finding a foothold between these polypoid masses which gradually spreads and becomes permanent. Confusion is very apt to be brought about when in cases of simple polyposis, a purulent infection occurs in an overlying sinus, particularly the frontal. This, however, is of little consequence as the treatment would be precisely the same at least in the beginning.

The diagnosis then of ethmoidal diseases while simple enough on paper, represents, at least in its incipiency, one of the most difficult and painstaking problems that ever confronts the sinuologist.

SYMPOSIUM ON THE ETHMOID.

CONSERVATIVE TREATMENT OF ETHMOIDITIS

SAMUEL IGLAUER, M. D.

CINCINNATI, OHIO

The arrangement of the ethmoid cells may be compared to that of a honey-comb. The cell walls are represented by the wax, and the secretion when present, is represented by the honey. Most of the ethmoid cells drain directly into the nose through small individual ostia, but occasionally there are cells which are connected with the nasal cavity only through a neighboring cell and its ostium. In the presence of infection, this anatomic arrangement leads to faulty drainage and ventilation of the cells with consequent retention of the secretions. The anatomic relations of the ethmoid labyrinth also prevent direct approach and treatment of the cells such as is possible in the antra, sphenoids and frontals. The para-nasal cells are probably involved in most cases of acute rhinitis, and undoubtedly the infection has a tendency to spontaneous recovery with the subsidence of the original infection.

PROPHYLAXIS

The prophylaxis of sinus infection is therefore the prophylaxis of acute upper respiratory infection. In our larger cities, two special factors promote the spread of respiratory disease, namely: crowding of the population and pollution of the atmosphere by dust and smoke. It has been shown, for example, that in Chicago, the amount of sunshine has been reduced 12 per cent in the last five years.[1] "The Department of health which has been making observations since early last winter finds that only half of the sunlight with its curative ultra-violet rays which we find on the tops of buildings reaches the surface of the streets." During those days in which there is little of the ultra violet light, cases of pneumonia increase as well as the number of deaths.

To the individual who can afford it, winter bathing in artificial sunlight increases his resistance to infection, but for the vast majority of city dwellers, smoke abatement is becoming a necessity for the prevention of respiratory diseases, especially influenza, pneumonia and tuberculosis. Patients who suffer each winter with sinus infection are greatly benefited by spending the sunless months in southern climes, such as Florida, Arizona, New Mexico and California.

Both Dean[2] and Shurley[3] have emphasized the importance of diet as a factor in para-nasal disease. To quote Dean, "the second most important thing in the treatment of para-nasal disease in infants and young children is diet." Deficiency in vitamin A seems to invite infection. Fenton[4] advises the avoidance of swimming and diving in patients predisposed to sinus infections. The removal of obstructing adenoids in children by restoring the free nasal respiration with its tidal flow, not only prevents, but often cures sinus infections. Children should be taught the proper technic of blowing the nose.

Patients who suffer from hay fever or vaso-motor rhinitis of allergic origin should be treated for these underlying conditions rather than for the secondary ethmoiditis which may be present in some of these cases.

Syphilis and nasal diphtheria should be excluded before undertaking local treatment.

Treatment

We are called upon to treat ethmoiditis in both the acute, the subacute and the chronic stages. The treatment of acute ethmoiditis is chiefly symptomatic; the objects being to relieve the pain and headache, to restore the patency of the nasal passages; and to favor the discharge of the secretions. For the headache, capsules containing codein and pyramidon are very efficacious. Some patients are relieved by aspirin which also promotes sweating, but which may increase the cerebral congestion. External heat from ordinary lamps or from infra-red rays tends to relieve the pain and fullness in the head. Sometimes ice compresses are more satisfactory.

The use of ephedrine preparations, preferably by a spray, tends to relieve the congestion and edema of the mucosa, and favors ventilation and drainage of the cells. It is a good plan to spray the nose, then wait for a few minutes until the anterior

portions of the nasal passages open and then immediately repeat the spraying to open the posterior portions. Suprarenal extract, if employed, should be very dilute (1:10,000). To this cocaine may be added to give a 1 per cent solution. The cocaine-adrenalin spray should be used for a few days only. A recent preparation, "Adrephine," a combination of adrenalin and ephedrin, is said to prolong the astringent effect produced by either drug when used alone.

Patients are greatly benefited by the use of the suction pump. A nasal tip is inserted into one nostril, the other is closed with the finger and suction is applied while the patient shuts off the oro-pharynx by phonating (K) or by swallowing. As I have shown in a previous paper (5) suction, to be efficacious, must be interrupted repeatedly to allow air to enter the cells and force out the secretion. The negative pressure should be as strong as the patient can tolerate. For home treatment, the rubber suction bulb and glass tip of the Sondermann should be prescribed. It can often be used in children as well as adults. Following suction treatment gentle Politzerisation may be employed.

After the very acute stage has subsided, the condition is improved by packing the middle meatus with colloidal silver solutions applied on cotton. I have found Hydrosol Argentium ("Collene") .05 per cent, very satisfactory for this purpose. It may also be used as a spray and does not irritate the mucosa or stain the linen. Argyrol, Protargol or Silvol tampons may also be employed. This treatment favors osmosis and probably reduces the infection.

If the secretions become thick and tenacious, irrigations with warm isotonic salt solution may be used in adults. In children, only small amounts of fluid can be introduced into the nose without endangering the middle ear, unless the child is held in the prone position with the face down and beyond the end of the table. The development of displacement irrigation by Proetz[6] [7] has constituted a distinct advance in both the diagnosis and treatment of sinus disease. By this procedure, it is possible to introduce fluids into otherwise inaccessible ethmoid cells, and to free them of their retained secretions.

In the conservative treatment of subacute and chronic ethmoiditis, as well as in the treatment of unsuccessful post-operative cases, we may employ many of the procedures mentioned

above, including suction, irrigation, displacement irrigation, and nasal tamponade.

Cases associated with crust formation are benefited by home treatment with an atomizer containing some bland oil to which Coryfin and a small quantity of iodine has been added.

It frequently becomes necessary to infract the middle turbinate in order to expose the ethmoid capsule for inspection and treatment. Infraction is usually accomplished with the long bivalve Killian speculum. I have found that the luxation of the middle turbinate is easily accomplished by means of a thin Freer elevator inserted under the turbinate.

Lederer[8] advocates the use of the electro-cautery in the treatment of recurrent polyposis without suppuration. He reports a series of more than four hundred cases without any fatalities. Others have refrained from this practice fearing meningitis as a possible complication of this procedure. The use of radium in the treatment of polypoid ethmoiditis is still in the experimental stage, although reports seem favorable.[9]

The correction of a high deviation of the septum, as well as the removal of hyperplastic mucous membrane and polypi might be considered under the subject of conservative treatment of ethmoid disease, but properly belong to the next two papers of this symposium which deal with the surgery of ethmoiditis.

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SYMPOSIUM ON THE ETHMOID.

RADICAL TREATMENT OF THE ETHMOID—INTRANASAL

RALPH A. FENTON, M. D.*

PORTLAND, OREGON

Approach through the narrow threshold of the nostril should impose a certain caution upon the operator, and his indication for operation should be an ethmoiditis limited to accessible intranasal cells. Contraindications of a general nature include disease which may be implanted in or become localized in the operative field, such as tuberculosis and syphilis. Disease affecting metabolism, such as diabetes or chronic nephritis, especially when associated with hypertension; severe anemia, especially with relative lymphocytosis; hemophilia, or any prolongation of bleeding or clotting time from nonhereditary causes, may all forbid or at least delay resort to radical intranasal surgery. Local contraindications include narrowed passageways, whether the small nostril of childhood, or the acquired atresia of healed septal abscess or other severe loss of substance in the vestibule and upper septum. Orbital or intracranial complications of ethmoid suppuration forbid intranasal work, except as a step in adequate external operations. If a coexistent suppurative process in the homolateral frontal or maxillary requires radical external operation, naturally the ethmoid may best be reached at the same time, externally.

Allergic hyperplasias affecting the ethmoid are not in our experience suitable for radical surgery, unless secondary stasis of exudates has led to purulent invasion. Such cases repay conservative management.

Freedom from external scarring and suturing is an obvious advantage, and no disturbance of the orbital contents, save occasional ecchymosis, is likely. Escape from the annoying psychic effect of transient diplopia such as may readily occur in the external orbital route is likewise a distinct advantage.

Most important is the fact that where time permits, intranasal surgery may be accomplished in several stages, permitting

*(From the Department of Otolaryngology, University of Oregon Medical School, Portland.)

meanwhile careful study of the ethmoid. Limited areas of the ethmoid may thus be reached, leaving other parts relatively undisturbed. This conservative tendency is gaining acceptance within recent years, in contrast with the very radical intranasal surgery of ten years ago.

Accessibility of the agger nasi and of middle turbinate cells is obviously greater by the intranasal route. (Fig. 1.) The field of vision of the operator is, however, at first rather strictly limited to the anterior halves of the inferior and middle turbinates, the agger nasi, and the nasal septum. Anything beyond, such as the bulla, hiatus and infundibulum, requires extensive shrinkage and often preliminary septal or turbinal work. Extranasal cells—in the orbital roof, frontal bullæ, between orbit and antrum, lateral to the sphenoid[1] are utterly inaccessible to intranasal procedures.

Danger of getting lost in the posterior ethmoid, if the head of the patient be turned up from the horizontal,[2] has been a very real peril in all procedures where the ends of instruments are out of sight of the operator. Very slight anomalies in this complex grouping of dural prolongations, olfactory and visual nerves, and

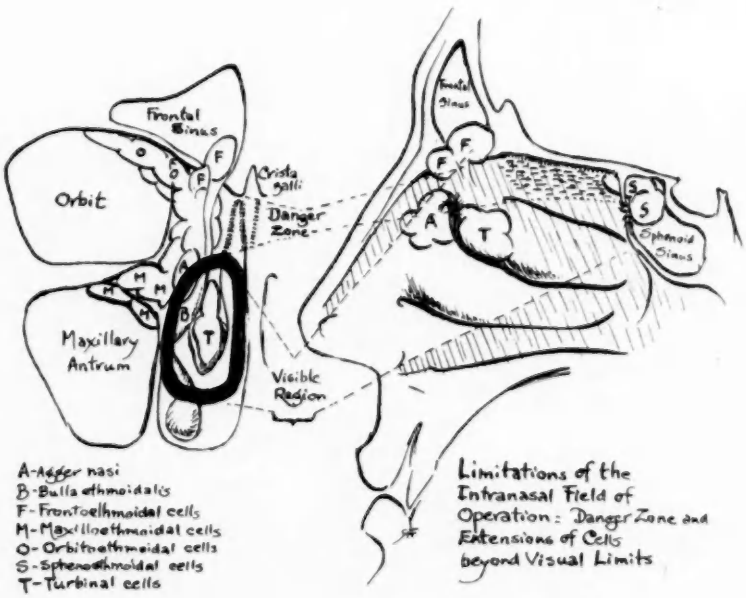

Fig. 1

blood vessels, may lead to complications varying from retrobular hemorrhage to optic neuritis or meningitis. Special training based upon dissection, cadaver work, and operative experience under the eye of a competent teacher, are needed before intranasal ethmoid work should be essayed. Such is the opinion of Luc,[3] Tilley,[4] Hajek,[5] Mosher,[2] Skillern,[6] Sluder[7] and many other distinguished clinicians.[8 9 10 11 12 13 14] Competence may not be acquired in this field by reading or by study of drawings. Depth perception must be accurate and important landmarks must be recognized, in a field likely to be dark, narrow and bloody.

Appropriate preliminary resection must be done if access to the ethmoid is hindered by high septal thickening with or without deviation (Fig. 2.) Such resection must often precede the equally necessary and desirable infraction of the middle turbinate. Also occlusive masses of anterior polypi should be removed, as far up as possible, with traction of the snare on their pedicles. Choanal polypi are almost always from the maxillary antrum, but may interfere with access to posterior cells. The latter may, like the antrum, be full of edematous or hyperplastic mucosa, but

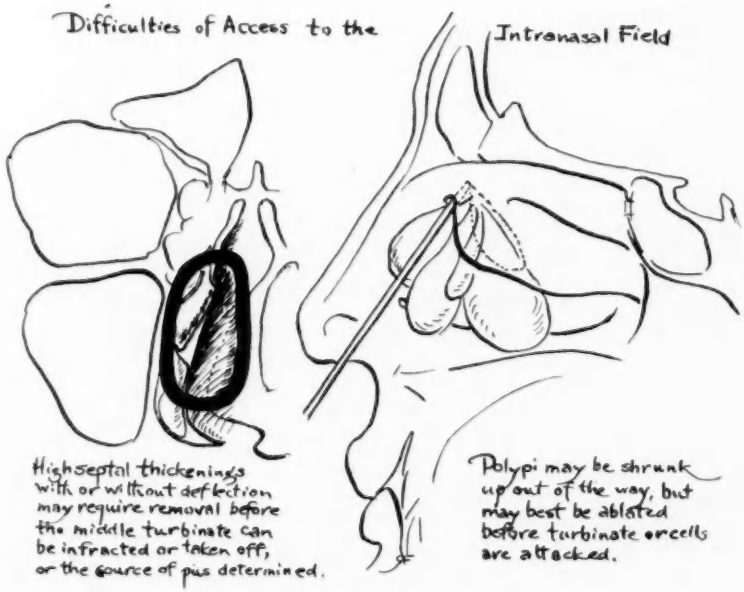
Difficulties of Access to the Intranasal Field
High septal thickenings with or without deflection may require removal before the middle turbinate can be infracted or taken off, or the source of pus determined.
Polypi may be shrunk up out of the way, but may best be ablated before turbinate or cells are attacked.

Fig. 2

almost never originate pendent polyps. The bulbous-tipped middle turbinate which contains a cell must usually be resected.

An effort should be made by studying stereoscopic radiographs to determine whether extranasal extensions of the ethmoid are present. Ocular symptoms—transitory dimness of vision, flickering photophores, paracentral scotomata, reduction of red-green sensitiveness—suggest radiographs in the positions of Rhese or White, to show possible encroachment upon the optic nerve by a posterior ethmoid cell. Lipilodol after ephedrin, by the suction technique of Proetz, has not helped us in anterior cells, though it is very useful occasionally in the posterior cells, as in the sphenoid.

Location of discharge should be accurately decided before the type of operation is selected. The ostia cannot be seen; but (having ruled out frontal and maxillary involvement) long cotton tampons may be packed along under the shrunken middle turbinate, others in the superior meatus back to the sphenoid, and left in place from ten to forty minutes. Inspection will usually show secretion opposite the suspected cells, particularly if some dark silver-protein solution has been used on the cotton tampon for contrast.

The objects of intranasal radical operation, which may best be a succession of relatively conservative procedures, are three: first, *to secure aeration*, with consequent shrinkage and retraction of exposed hyperplastic mucosa; second, *to secure drainage*, where liquid exudates are actually present; and third, *to avoid invasion of unaffected cellular structures* during the operation if possible.[6]

Our own preference is for the scopolamin-morphin preparation, with local anesthesia. We prefer this to general anesthesia, which increases hemorrhage during operation and recovery, and subjects the patient to danger of pulmonary involvement from inhaled septic material and blood. Prior to entering the agger region, submucous injection of 1 per cent novocain with a very small amount of adrenalin may be made. For rapid surface action we use 10 per cent cocain with a little 1/1000 adrenalin, but where speed is no object we prefer 5 per cent butyn, also with adrenalin, and have had so far no ill effects from it. These are applied in the usual fashion with two small initial pledgets high up to block the sphenopalatine and anterior ethmoid region; then long cotton tampons barely moistened by the solution are placed

under the middle turbinate, about the bulla, and above the middle turbinate from the choanal rim forward about the superior turbinate. We do not like cocain "mud," although many excellent operators use it constantly. Above all, excess solution must not be allowed to run into the pharynx. We have found that the scopolamin-morphin preparation abolishes anxiety and its corollary of fainting or collapse, often ascribed to the local anesthetic. The bother of interruption for clearing the pharynx is avoided by packing a large cotton wad or rolled gauze strip into the choana before extensive procedures are started.

Intranasal ethmoid procedures, then, may be grouped as follows:

Improved methods of access—septal resection, partial middle turbinectomy, infraction of the middle turbinate, removal of polyps;

Invasion of the anterior cells—often associated with frontal involvement; operations on the agger nasi, uncinate and bulla (Coffin,[15] Hajek,[5] Halle,[16] Skillern,[6] Mosher[2]);

Invasion of the anterior cells carried back through the posterior cells without removal of the middle turbinate (Pratt[17]);

Invasion of the anterior cells carried back through the posterior cells with more or less complete turbinectomy (Mosher[2], Tilley,[4] Halle,[16] Skillern,[18] Coffin,[15] Hajek[19]), or from behind forward (Moure-Portmann[20]);

Bloc removal of the ethmoidal mass with turbinate attached (Ballenger,[21] Sluder[7]).

We have attempted to visualize simultaneously the picture presented to the operator as he looks up into the shrunken nose toward the middle meatus, with a lateral diagram of each operation. In this way we may see very quickly how much work has to be carried on beyond visual control, in many of these standard procedures.

We find the quick snip of scissors (Fig. 3) preferable to the crunch of forceps (Fig. 4) or the crackling of a push-chisel, for the initial cut of middle turbinectomy. A rachet-pattern pistol-grip snare with stylet, which may be reset, is better than those which pull a long wire loop around a corner to get stuck in the canula. Twisting off the middle turbinate with Luc's forceps now seems unnecessarily brutal, (Fig. 5) though excellent access is thus afforded to the bulla and other cells, which may be

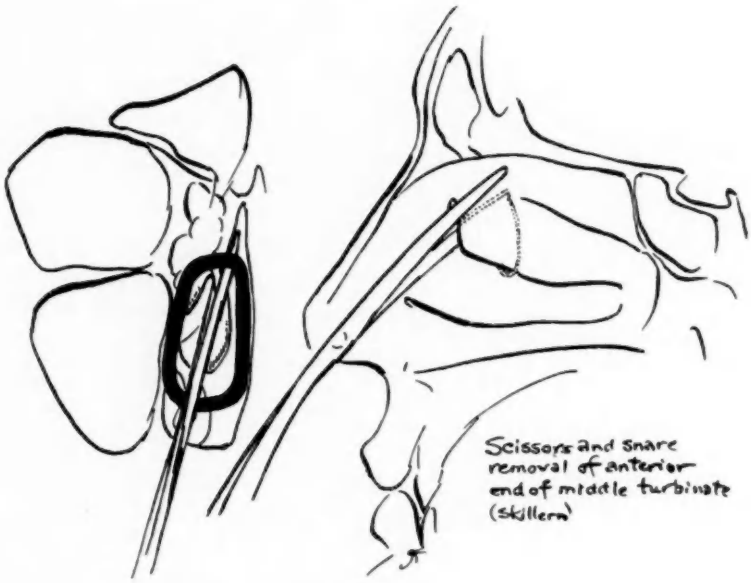
Scissors and snare
removal of anterior
end of middle turbinate
(Skillern)

Fig. 3

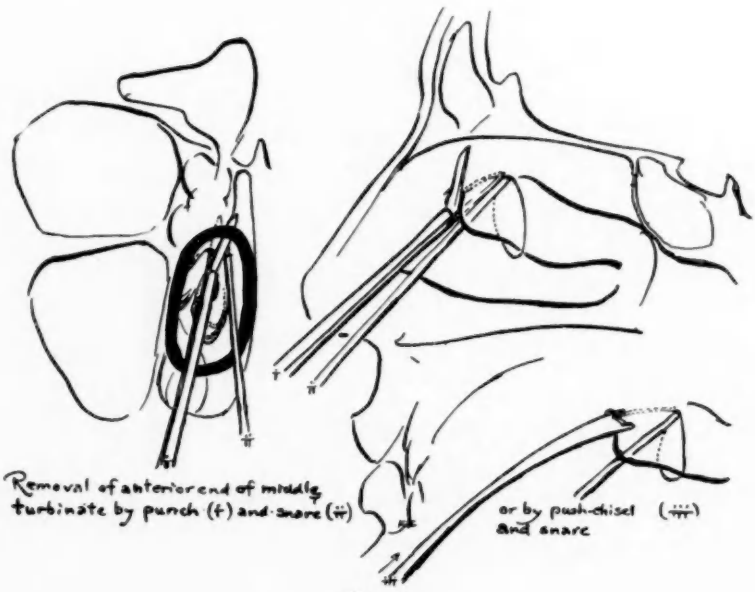
Removal of anterior end of middle
turbinate by punch (†) and snare (‡)
or by push-chisel (⁂)
and snare

Fig. 4

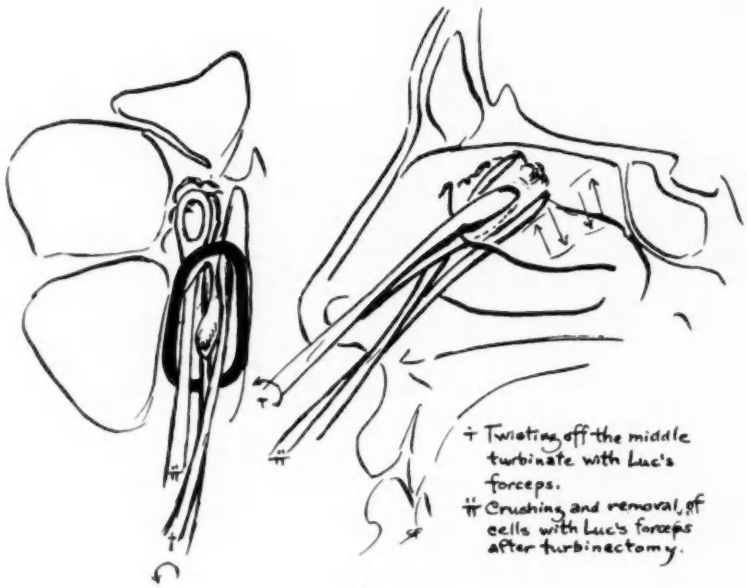
† Twisting off the middle turbinate with Luc's forceps.
‡ Crushing and removal of cells with Luc's forceps after turbinectomy.

Fig. 5

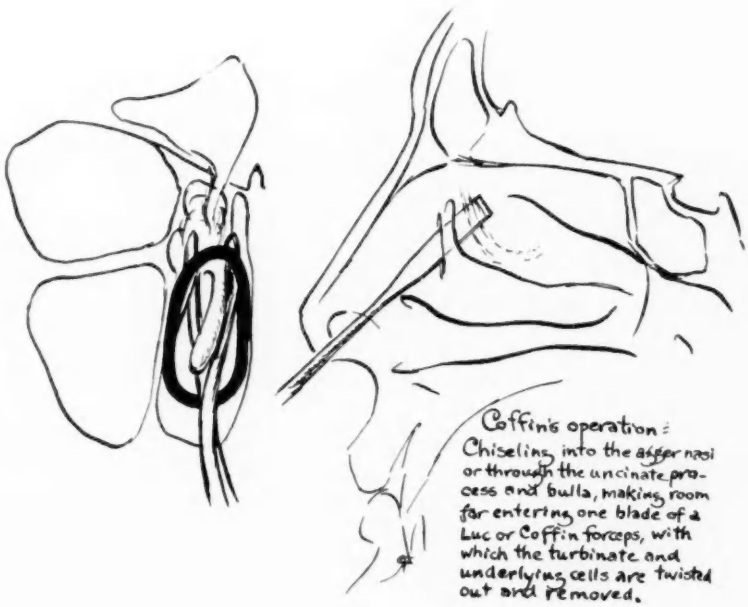
Coffin's operation:
Chiseling into the agger nasi or through the uncinate process and bulla, making room for entering one blade of a Luc or Coffin forceps, with which the turbinate and underlying cells are twisted out and removed.

Fig. 6

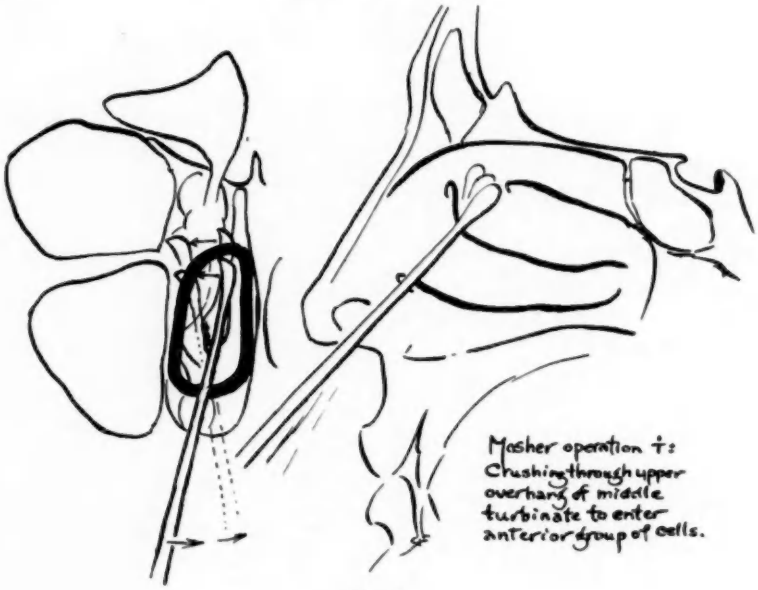

Fig. 7

smashed up progressively in the broad and relatively safe jaws of these forceps.[3] [22]

Coffin[15] [28] enters the agger cell and transfixes the uncinate with a flat chisel (Fig. 6) thus making a path outside the bulla for one blade of his forceps. The other blade lies internal to the middle turbinate, and by a twisting closure the anterior mass is broken from place, giving access for such deeper removal as may prove necessary.

The Mosher [2] [4] [9] [10] [22] [26] operation smashes through the anterior overhang of the middle turbinate (Fig. 7) outward into the anterior group of cells, using his special curette. Thence, with a pawing motion backward and downward, he tears down the remaining septa and the posterior cells as far as the sphenoid, warning meanwhile against backward displacement of the head (Fig. 8). The back part of the middle turbinate has remained as a landmark, but is at length removed by forceps and snare, except at the posterior extremity. Cells in front of the hiatus are curetted forward. The square ended "spade" curette is safer in most hands than the original Mosher instrument.

Moure's operation[12] [20] reverses the Mosher technique (Fig. 9) using a curette almost as large as the Luc forceps, mounted in

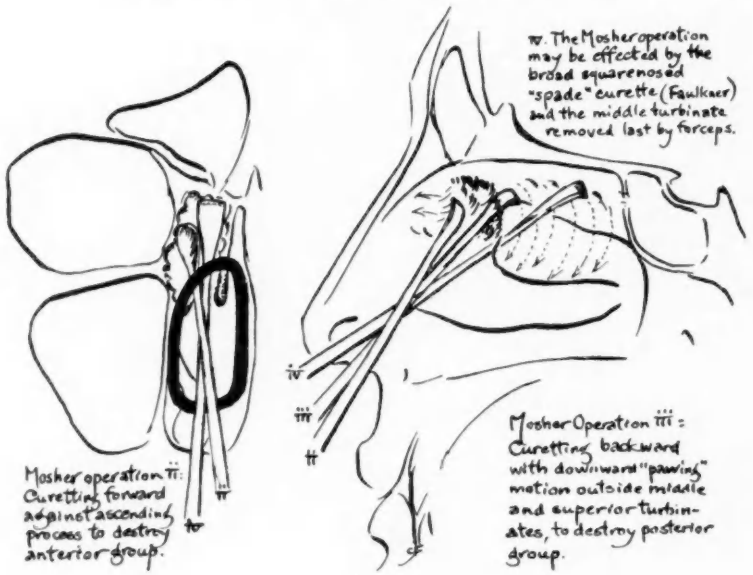
iv. The Mosher operation may be effected by the broad squarenosed "spade" curette (Faulkner) and the middle turbinate removed last by forceps.
Mosher operation ii: Curetting forward against ascending process to destroy anterior group.
Mosher Operation iii: Curetting backward with downward "pawing" motion outside middle and superior turbinates, to destroy posterior group.

Fig. 8

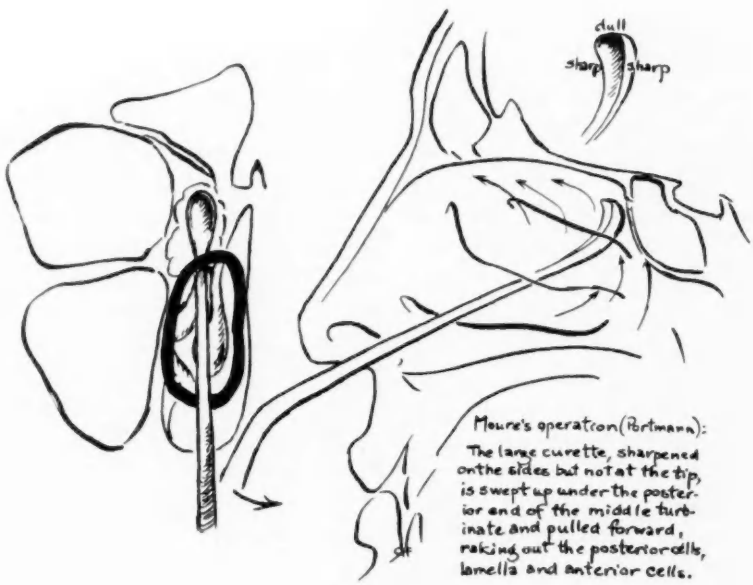
dull
sharp
sharp
Moure's operation (Portmann):
The large curette, sharpened on the sides but not at the tip, is swept up under the posterior end of the middle turbinate and pulled forward, raking out the posterior cells, lamella and anterior cells.

Fig. 9

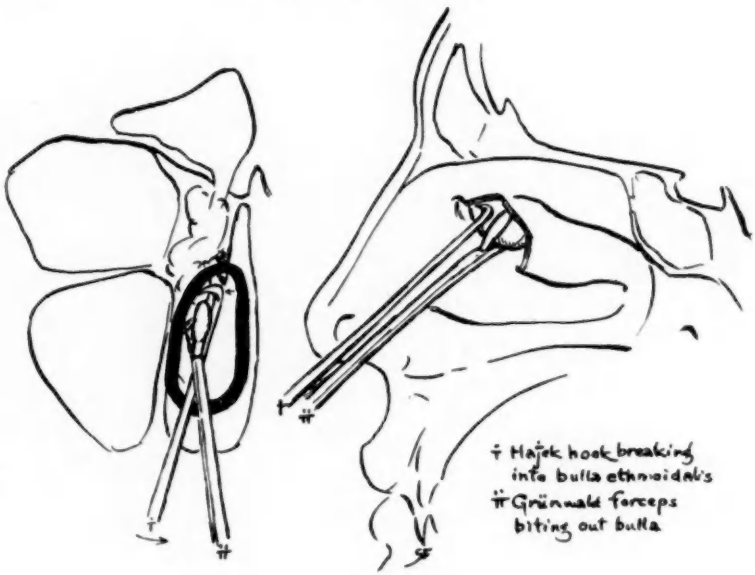

Fig. 10

a double curved handle. The spoon of this curette is sharpened sidewise, but not on the rounded end. This formidable instrument is entered under the back end of the middle turbinate, swept up along the sphenoid wall and forward to the agger nasi, ripping out cellular structures from behind forward as the handle is depressed.

After removal of the anterior end of the middle turbinate, Hajek[5 23] is accustomed to open the bulla with his ethmoid hook. (Fig. 10). Grunwald's forceps are now used to bite away cells and tags and to open other anterior cells. This hook is also a useful instrument for raking down the superior turbinate and posterior ethmoid group, (Fig. 11) as far back as the sphenoid[18 19] Its beak should not be long enough to reach through to the orbital wall.

Halle's technique,[16 24 27] primarily designed to give a broad entrance into the frontal (Fig. 12) by rolling down a mucoperiosteal flap from the region of the agger nasi and ascending process, gives excellent access also to the anterior ethmoid. Chiseling away the agger nasi and uncinate, Halle infracts the middle turbinate and bites away the ethmoid cells as far back as necessary under the intact turbinate. The flap is lastly turned back over the

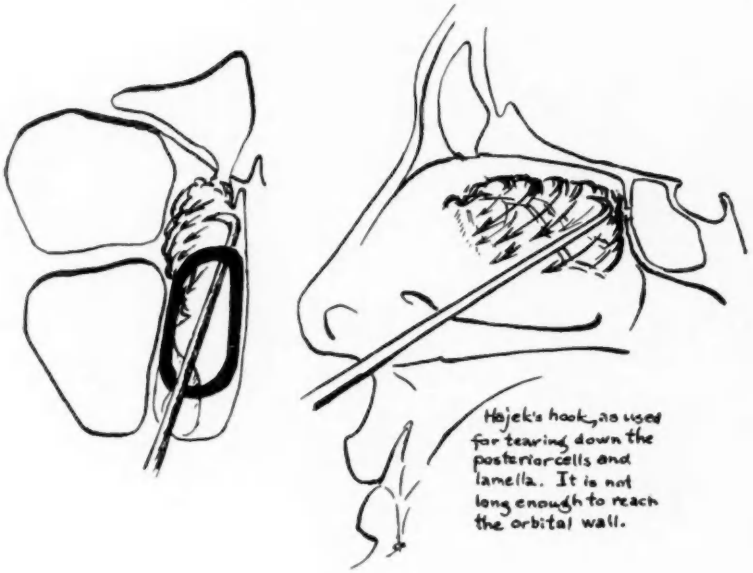
Hajek's hook, as used for tearing down the posterior cells and lamella. It is not long enough to reach the orbital wall.

Fig. 11

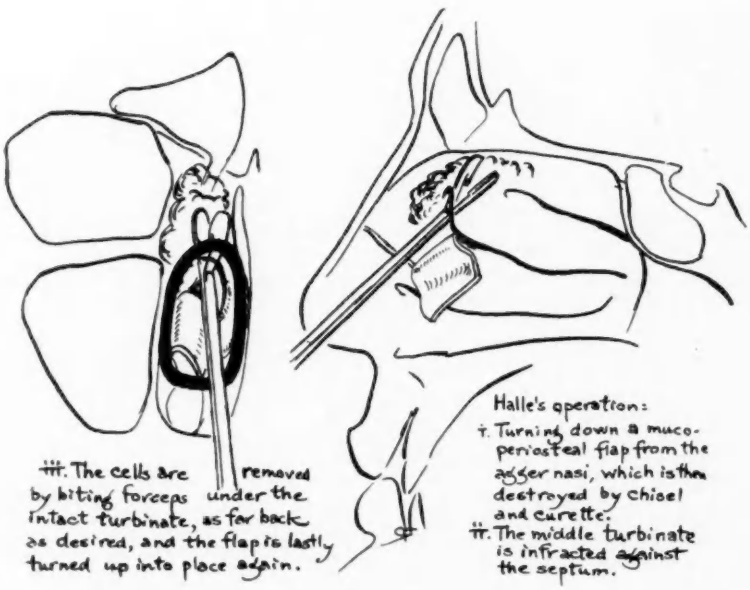
iii. The cells are removed by biting forceps under the intact turbinate, as far back as desired, and the flap is lastly turned up into place again.
Halle's operation:
i. Turning down a muco-periosteal flap from the agger nasi, which is then destroyed by chisel and curette.
ii. The middle turbinate is infracted against the septum.

Fig. 12

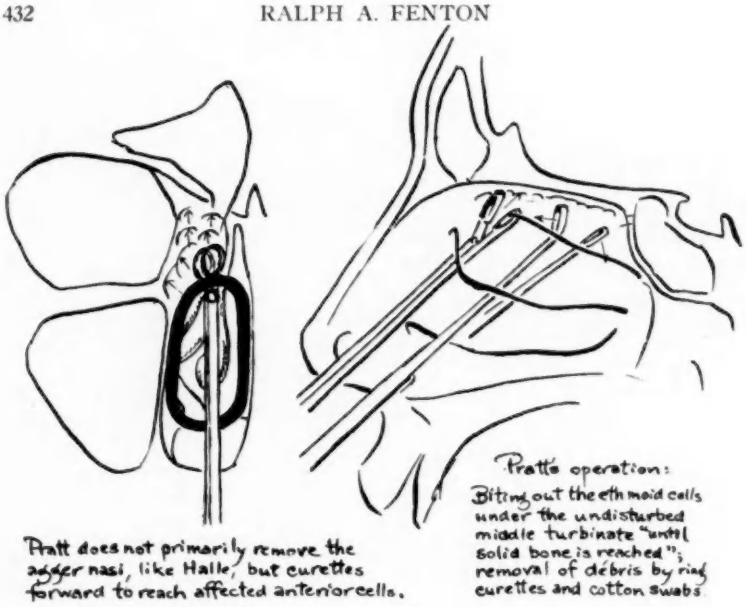
Pratt does not primarily remove the agger nasi, like Halle, but curettes forward to reach affected anterior cells.
Pratt's operation:
Biting out the ethmoid cells under the undisturbed middle turbinate "until solid bone is reached"; removal of debris by ring curettes and cotton swabs.

Fig. 13

denuded agger region. Pratt[17] does not remove the agger primarily, but curettes forward to reach anterior cells (Fig. 13). He preserves the turbinate at all hazards, biting out cellular structures by touch "until solid bone is reached," and removing debris by special large ring curettes and cotton swabs.

The earliest bloc removal, (Fig. 14) that of Ballenger,[21] was effected by a large sickle-bladed knife which has a right or left handed straight blade at its distal end, set at right angles and long enough to reach from the orbital wall almost to the septum. This powerful instrument is slipped up behind the bulla and under the middle turbinate, and as its handle is depressed the posterior cells, lamella and anterior cells are rocked loose. The entire ethmoid capsule with attached turbinates is thus sliced free and drops down for removal. Carelessness, or unperceived anatomic irregularities, have brought about invasion of the dura, orbit, or top of the septum during this operation.

Sluder's operation[7, 26] required anterior severance under the middle turbinate (Fig. 15) by his short and powerful right-angle knife; he then slid the back of the same knife far along the cribriform plate and turned its square cutting tip outward, dragging it forward to connect with the previous subturbinal cut. The

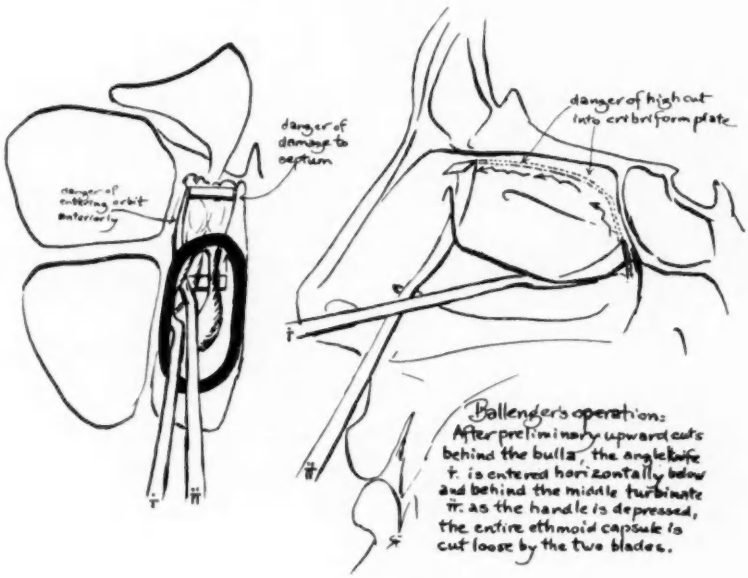
danger of damage to septum
danger of cutting orbit anteriorly
danger of high cut into cribriform plate
Ballenger's operation:
After preliminary upward cuts behind the bulla, the angle knife i. is entered horizontally below and behind the middle turbinate ii. as the handle is depressed, the entire ethmoid capsule is cut loose by the two blades.

Fig. 14

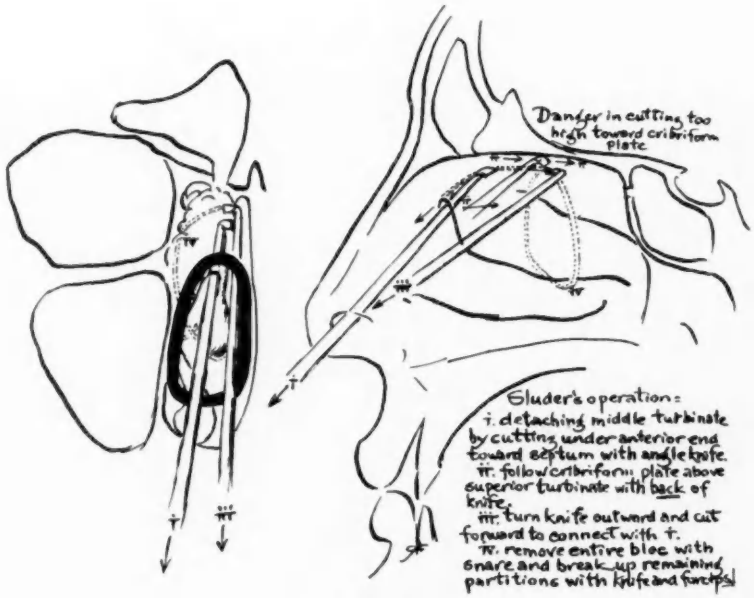
Danger in cutting too high toward cribriform plate
Gluder's operation:
i. detaching middle turbinate by cutting under anterior end toward septum with angle knife.
ii. follow cribriform plate above superior turbinate with back of knife.
iii. turn knife outward and cut forward to connect with i.
iv. remove entire bloc with snare and break up remaining partitions with knife and forceps.

Fig. 15

turbinate and its underlying cells, freed if necessary by further lateral cuts through the cell walls, were removed by a snare placed as far back in the incision as possible. Partitions thus laid bare were smoothed by knife and forceps. As with Ballenger's operation, this bloc procedure has caused a good deal of trouble at the hands of unskilled operators.

What shall be the procedure of choice for the average operator? Men from various schools, trained by various teachers, will give various answers, of course. To invite discussion, and without any intention of dogmatism, we may suggest the following steps:

In hyperplastic cases:

a. *High septal resection:* If speed is not essential, *wait*[6] awhile to see if room enough has not been gained;

b. *Removal of the anterior end of the middle turbinate; removal of polypi,* as completely as possible: Again, *wait*[6] if time permits;

c. *Remove agger and uncinate* by chisel;

d. *Break down bulla* with curette and forceps;

e. *Curette away affected anterior cells;*

f. *Break into affected posterior cells* with hook, smoothing up with forceps.

The last four steps may be carried out as one operation, but the piecemeal method of delaying the early steps will be found conservative of uninvolved areas, and less likely to give rise to secondary infection therein—so-called recurrences—than the extensive one-stage operations.

General suppurative changes involving the entire ethmoid capsule, with or without polypoid degeneration, call for complete removal, and nothing is gained by delay.

In complete suppurative cases, therefore:

a. *High septal resection* (if needed for access): The septum should be entered from the opposite side, if done simultaneously with the ethmoid;

b. *Middle turbinectomy* with scissors and snare;

c. *Remove uncinate* by chisel, or *destroy bulla* by spade curette, depending on the anatomical conformation;

d. *Curette and punch away cells* to anterior wall of sphenoid working always back and down, *never outward or upward.*

Choice of instruments should be limited to those of rather broad rounded outlines, (Fig. 16) to avoid inadvertent damage

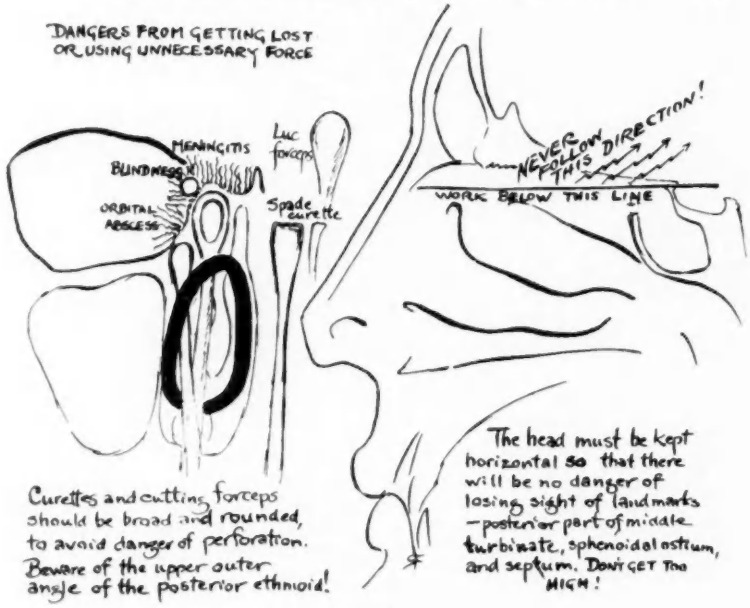
DANGERS FROM GETTING LOST OR USING UNNECESSARY FORCE
MENINGITIS
BLINDNESS
ORBITAL ABSCESS
Luc forceps
Spade curette
NEVER FOLLOW THIS DIRECTION!
WORK BELOW THIS LINE
Curettes and cutting forceps should be broad and rounded, to avoid danger of perforation. Beware of the upper outer angle of the posterior ethmoid!
The head must be kept horizontal so that there will be no danger of losing sight of landmarks — posterior part of middle turbinate, sphenoidal ostium, and septum. Don't get too HIGH!

Fig. 16

to vital structures. The danger zone at the upper outer edge of the posterior ethmoid must never be forgotten. Mosher's warning that the patient must keep his head level instead of tipping back (withdrawing from the operator) is of vital importance during all intranasal work; otherwise it is easy to lose sight of important landmarks, especially the posterior end of the middle turbinate. Slashing indiscriminately up into the olfactory nerve sheaths may cause fatal damage.

Immediate after-treatment of intranasal ethmoid operations should be limited to hemostasis—for oozing, light cotton tampons with thromboplastin, coagulen, etc., for a few minutes. The wound may be swabbed with 2 per cent mercurochrome, 3 per cent iodine, or compound tincture of benzoin. Packing should never be used.

Subsequent treatment should be of the slightest. Wound exudates should not be disturbed; clots and scabs are best left alone. There should be no swabbing, no spraying, no blowing of the nose, and as little postnasal "hawking" as possible. A few drops of gomenol ½ per cent in plain paraffin oil, or a small quantity of boric-lanolin (borofax) ointment, will allay local irritation and facilitate separation of crusts in a few days.

The normal process of healing should not be disturbed by fussy attempts to "clean up the wound." Annoying bleeding and secondary infection, with prolonged convalescence, may readily occur.

Within the strict limitations of its indications, intranasal surgery of the ethmoid has been found highly satisfactory; but excessively radical methods carried out blindly through the narrow nasal pathway have not stood the test of matured experience. No better conclusion can be made to this estimate of the present status of these procedures than these words of Herbert Tilley[30] ". . . It must be stated emphatically that many of the local and general symptoms . . . may be practically cured by operations which involve little risk in skilled hands, even though the results may not survive the searching criticism of the surgical idealist. Hence it is incumbent on those who deal with these matters—(1) to make an accurate diagnosis of the extent of the lesion; (2) to determine how far the condition probably threatens the health, happiness, and even the life of the patient; and (3), if an operation be decided upon, to bring to it a greater combination of anatomical knowledge, caution, and manipulative skill than is demanded in any other form of nasal surgery."

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SYMPOSIUM ON THE ETHMOID.

ETHMOIDAL SINUSITIS, EXTERNAL RADICAL APPROACH

R. C. Lynch, M. D.,

NEW ORLEANS, LA.

The indications for an external approach to the Ethmoid district may be roughly divided into two general classes.

First, the acute manifestations not only of inflammation and suppuration in this area alone, but more properly because of the extension of this inflammatory process to or towards adjourning important structures, Orbit and Brain, or on account of some specific accidental injury of such character as to force invasion of this district for the relief or repair of defects or the arrest of complications.

Secondly, as a last resort to clear up a previously persistent pathology which has resisted all methods of less violent approach or for the eradication of this district as a focus of infection in the presence of a secondary manifestation.

It has always been my experience that the surgery of the first group is not only nearly always emergency in character but the surgery here in the midst of acute violent septic bacterial invasion is of a type which does that only that will give free vent to pus accumulation, produce sufficient decompression principally on the orbital contents and maintain free drainage both externally and through the nose until the symptoms of the acute storm have subsided and subsequently repair the drainage in such a way as to prevent a recurrence of another such storm.

What I have had to do under these circumstances was to incise along the rim of the orbit and lateral nasal wall from the supra-orbital foramin to the inferior border of the nasal process of the superior maxilla, through the periosteum to the bone, peel back the periosteum along this area on its lower border only into the orbital cavity to and beyond its point of rupture from the Ethmoid or frontal into the orbit. Open the frontal at the T suture made by the nasal process of the superior maxilla nasal

and frontal bones, take away the nasal process of the superior maxilla and that portion of the ethmo orbital wall back to the point of invasion. This not only relieves the orbit of further invasion but is a good decompression for the already inflamed orbital contents drains the frontal through a large opening both into the nose and out through the incision using rubber tubes usually for the purpose and do not suture the wound.

In three of seven such invasions it was necessary to incise the orbital periosteum from before backward to facilitate drainage of pus from this area and this too in my experience is the most satisfactory method of draining for orbital abscesses of this kind. My slogan is to get in, do that only which is necessary to give free drainage and then get out quick and fight the constitutional manifestations of the storm by the methods which are familiar to you all.

Six of seven lives, five of six eyes stand to the credit of the above outline so far, one case died of meningitis as usual.

The external approach to the ethmoid spenoid frontal area because of a long persisting symptom producing chronic pathology is a far different process. In the first place there are those who think this the only pathway to approach the problem and all cases of ethmoiditis of a chronic nature must be treated by this route. I do not belong to this school, but feel certain that the external approach will bring about an ideal result, when the methods proposed by Dr. Iglauer and Dr. Fenton fail to accomplish the desired end. Because of anatomical variations which are many, of pathological changes which will not yield to drainage and ventilation and of the mechanical impossibilities of complete exenteration or evisceration by the intra nasal route, there will always be that number or group, who must finally submit to the external approach if they expect to be symptom free. Personally I have never seen a case of chronic suppurative ethmoiditis per se, with no pathology in the adjoining cavities—nor have I seen a chronic hyperplastic ethmoiditis all alone, so that my discussion of this subject must necessarily include the frontal and sphenoid and antrum as well. If either of the above conditions should exist I am quite sure the methods advocated by Dr. Iglauer first or Dr. Fenton next would cure them and I would never be called upon to approach the part from an external route.

The problem presents itself then after this fashion; medicine local and general, including diet, hygiene and climate have failed—intra-nasal surgery no matter by what method or in whose hands have failed to bring relief of pain, discharge or remove the focus of infection. It is then that the external approach is the only one left and this must be planned in such a way as to most effectively remove every vestige of pathological tissue, otherwise this too will be a failure. I have never agreed to nor can I see the rationale of thinking that given a marked pathological change in the mucous membrane of a sinus sufficient to resist medicinal and intranasal surgical methods, that only a part of this membrane is affected and that operation based on this theory that only the microscopically diseased membrane should be removed.

Some eyes may be very sharp but the unaided eye cannot see either microscopical changes in the tissue nor its bacterial content. It is more or less common experience for instance, to submit the tissue thus removed from a sinus to the Pathologist, who grinds this tissue and macerates it and cultures from this macerated tissue will many times show a hæmolytic streptoccous. Since this has been observed so often as to be a fact, I am convinced that the only fundamental principal to be considered is that which has for its aim the removal of every vestige of pathological tissue.

Here is neither time nor place to preach conservatism if one is to expect a cure—and likewise I cannot see how conservative modifications of the various radical procedures proposed can be expected to result in a cure which means that the patient is not only free from pain and discharge, but must be free from the focus not only for present states, but in anticipation of what may come if this is not the case. Operative procedures which clear out the ethmoid completely but leave the frontal sinus to drain through a more or less small opening is a mistake, even the reporter of such a technique admits that the frontal must be sounded for a month and admits the necessity of re-entry because of continued symptoms and pathological manifestations. Also those who enter the frontal and remove only the neighboring ethmoid cells, come to grief for the same reason. If the antrum or sphenoid are neglected if diseased and they most usually are there will be sadness because of continued symptoms.

It was because of the above experiences, which I observed and waded through myself that I proposed that technique which has given me the best results in these cases. Since its presentation before The American Laryngological society in 1922 and before the Triological in 1924, I have had what I consider the best evidence that the proposition is correct. Men who have witnessed the operation and have themselves operated by duplicate technique, report to me and in the literature that their results are like mine. I have been trying to satisfy myself what happens to the operative area when healing is complete and there has elapsed sufficient time for the tissue to get set as it were, to its new order. Looking into these noses, watching the scar and observing the patients, I was convinced that the frontal was completely obliterated as an air cavity and was free from any secretive or absorbtive elements which was the ideal I have striven for. I will show you a group of X-ray plates which seems to me to indicate that at least the frontals are no longer air cavities, that the ethmoid, sphenoid and antrum no longer contain air or secret fluid. When this result is accomplished then only is the removal of the pathology complete and the desired end obtained.

My cases number now one hundred and forty-eight. In this number there are two deaths and no further complications to report that differ from the original published in the Transactions of the A. L. R. & O. Society for 1924. I am appending a description of the technique I follow, brought up to date. The minor changes are those only of simplification which time and experience have shown to be worth while.

If the antrum is diseased, and it usually is, then it is attacked first, doing a radical Caldwell Luc operation. If the middle turbinate is still in place it is removed and through the antral ethmoid area all ethmoid cells are attacked and cleaned out—also at this time the sphenoid is opened and drained, taking its anterior wall down as near the floor as possible and its membrane is very carefully removed at this time. I feel certain that the removal of all of the sphenoid lining can be done in a certain number of cases but this must be done with the greatest caution and regard for its anatomical relations and boundaries. An antranasal opening into the frontal can be done at the same sitting—this procedure will obviate the necessity for further external approach. If it does not clear up the situation, and it will not in a

certain proportion, then the external pan-sinus operation as proposed is carried out with care for all its details—as follows:

The principle of the attack is fashioned after Knapp's operation; the detail of the technique seems to be that which is ultimately responsible for the proper healing and final cure of the condition. The nasal cavity is prepared as for the nasal plastic in surgery at the present time—alkaline douches with through swabbing with 5 per cent Picric acid in alcohol which will not destroy surface epithelium but has sufficient penetrating power to be of marked antiseptic value; oral hygiene is likewise necessary and teeth, gums and recesses are covered with the same solution. A post nasal plug on the operated side is carefully placed and the threads are left long enough to facilitate its removal at the completion of the operation.

The skin surface is washed with alcohol and then ether is preference to benzine and solution of picric acid 5 per cent in alcohol applied broad and far of the field extending to the eyelash line on both sides. While the staining of the skin may be objectional it has not been found so in my cases, and we have noticed less skin infection. The eyebrow is carefully considered in this preparation but is unshaven and it is apparent that the best cosmetic results are through the unshaven eyebrow. The eye is carefully washed with a bland solution and the lids are sealed with two michel clips—these stay in place better and are cleaner and easier to apply and a cushion of gauze laid over for protection.

One cross cut is made at the upper inner angle of the incision—this is about the location of the T suture and is sufficient for proper suturing after operation. The incision is as for the Killian or Knapp procedure, but does not at first extend outwardly beyond the supraorbital notch until it is found by intrasinus measurement that this is necessary. We proceed gradually through the periosteum and when this is accomplished all vessels are tied off with the smallest of catgut to avoid the possibility of the forceps injuring the eye.

The periosteum is elevated only from the lower half of the incision. If the periosteum is elevated over the frontal plate of the frontal bone or represented by the upper half of the incision it is not likely to reapply itself to the bone again and in consequence there will develop either immediately or within three months a periostitis with elevation and a sequestrum of more or

less size will be cast off; this has happened on four or five occasions in my early sinus operations and I have been able to overcome this disturbance by adhering to this point. It will also protect the patient from the risk of an osteomyelitis gaining its start through this area especially when the periosteum does not reapply itself quickly as may happen either from too early blowing the nose or from post operative edema that follows in some of the cases.

In elevating the periosteum from the lower half of the incision, great care must be taken not to button-hole the orbital periosteum as this may be followed by disturbance of the orbital balance, but the remotest area of orbit sinus wall must come into view. In elevating the periosteum over the nasal process of the superior maxilla lower lateral edge of the nasal bone in the region of the upper portion of the lacrimal sac which is carefully dislocated and in the area of the superior oblique which is also dislocated, a sharp gouge is found to be the best instrument to use. A long submucous elevator exposes best the lacrimal and lamina papyracea of the ethmoid in most instances extending backward to within a few millimeters of the optic foramen. This is necessary to gain entrance to the post ethmoidal cell group and to give the widest field of approach to the anterior wall of the sphenoid. It must be remembered that the angle formed by the orbito-sinus wall or floor of the frontal sinus with its posterior or brain wall must be completely obliterated, and especially is this true at the external angle, for this is the area that most often harbors the infected mucous membrane which causes failure. When the common sinus orbit wall has been carefully removed the mucous membrane of the frontal sinus is carefully curetted away and again any remaining mucous membrane degenerated or not will conduce to failure. With the Coakley curets I have not met so far the sinus which will not permit of a most thorough curettage of the entire cavity. This done, a strip of gauze soaked in iodine is packed firmly into the cavity to prevent or guard against any infection entering this area from below and it will control bleeding very nicely.

Since Dr. Sewell presented his instruments for the retraction of the orbit and ligature of the ethmoidal and spheno-palatine arteries, I have not found it necessary to put in the catgut sutures for retention or retraction of the orbit. I find these retractors more adaptable to the necessities of the work and use them ex-

clusively now. With the removal of the nasal process of the superior maxilla, the lower lateral edge of the nasal bone, the lacrimal and the entire lamina papyracea of the ethmoid, there results a complete evisceration of the entire ethmoidal labyrinth since the middle turbinate has been previously removed and particular search is made for those orbital and sinus extensions of the ethmoid.

The roof cells of the ethmoid next receive our attention and these can be so comfortably cleaned out by direct vision without the least fear of injury to the cribriform area though sometimes I lay a stout probe along the roof of the nose through the nostril to act us a safety guide, the end of this probe resting in the sphenoid sinus to act again as a guide when its anterior wall is reached. The post ethmoid sphenoid cells standing plainly in view are broken down and every vestige of the mucous membrane is removed in this quarter. Finally the anterior wall of the sphenoid is removed completely; that means to its very limits and the lining membrane completely eviscerated. This is only possible by such an approach and even here I do not think it can be accomplished in every instance. In the groups of cases there is one where this was not done well and I have had some trouble in controlling a persistent discharge due to this fact. If the nose is cleaned and dried one can get the two angles of view for proper and comfortable orientation. The raw bone surfaces are carefully sponged with tincture of iodine and this is done as one of the first steps on the frontal plate of the frontal bone. It is not necessary to disturb the nasal spine of the frontal or to open this cancellous area to the wound because it is a fertile source for subsequent infection and will produce granulation tissue in abundance sufficient to interfere with drainage.

A drainage tube about ⅛ in. in diameter with one end cut on a long bevel is passed through the vestibule of the nose and lays in what was the area of the beginning infundibulum of the sinus. The iodine gauze is removed from the upper portion of the sinus cavity and the entire cavity is swabbed with iodine. If there has been bleeding from the post ethmoid sphenoid region a strip of idioform gauze dipped in tincture of benzoin compound is packed loosely to control this area. The frontal area does not bleed. Interrupted sutures of chromic catgut 00 are used to bring the subcutaneous tissues together but the needle point is not permitted to pass beneath the periosteum of the

upper half of the incision though the suture takes in the periosteum on the lower half of the incision and they are placed with due regard to the cross cut of the external incision.

The wound edges and neighboring field is carefully cleaned of all blood, using ether for this purpose, and the skin is brought together with metal clips using only enough tension to secure accurate approximation. No external drainage is necessary. The eye is now uncovered, carefully washed out again and a 20 per cent solution of fresh argyrol is instilled. The eye is left uncovered in the final dressing. I formerly used a quantity of vaseline and a large external dressing. Now I put on a small gauze pad cut the shape of the incision, leaving the eye exposed. The gauze pad is held in place by small adhesive strips. There is less swelling of the eye tissues and many times it is opened throughout the convalescense, the wound heals as well and the patient is more comfortable.

The gauze strip is removed from the nose in thirty-six hours and a sterile probe is passed through the rubber tube daily to insure its patulency. The rubber tube is left in place for three days as a rule when it is removed and the clips come out usually on the fifth or sixth day. After the removal of the rubber tube no dilator is used—this differs from the former recommendation to use a dilator. I find there is no necessity for this dilatation and it is disturbing to the healing process. No washing of the sinus or the nose is permitted and we feel the less the wound is interfered with after the operation the more rapid and perfect the healing. The discharge of course stops at once and does not recur. The healing is complete with a perfectly cicatrized nose as the result with no scab formation and no discomfort.

I have now followed this technique 148 times and my assistants, associates and friends have run this number up greatly. Our experiences agree and I take it that any technique which will give the same results in other hands than your own is the strongest recommendation that the principles and details are correct—and again I recommend it to you as the best.

Dr. Joseph Beck presented this paper.

DISCUSSION.

Dr. Eugene R. Lewis, Los Angeles: I had hoped that this symposium might present physiology, diagnosis from a broader viewpoint, and particularly treatment from the standpoint in which I have been most interested for many years—the combined surgical and non-surgical measures which, in my hands, have proved superior to the radical measures presented by Drs. Fenton and Lynch. My views are diametrically opposed to those expressed in so far as they are revealed by the statement that when ethmoid, sphenoid and antrum no longer contain air or secretion, the desired end is obtained. I realize that the speaker states clearly that he is referring to obstinate cases "which have resisted medicine, local and general, including diet, hygiene and climate," but even in these cases I have found it possible to attain results far superior to the condition he speaks of as the desired end. Restoration of normal conditions must always be the desired end. In certain cases this is manifestly impossible, but the nearest approach to this must still remain the goal of treatment for any individual case.

In my experience, persistent application over months and months of conservative measures which bring to bear every possible influence—surgical, medical, dietary, physiotherapeutic, supportive, stimulant, and, above all, time-consuming, frequently is attended with a degree of success that seems impossible at the outset.

In my opinion, remote factors of widely diverse nature enter into the etiology of the common infectious inflammations of the nasal structures, which I understand are referred to under the title of this symposium. Among these may be mentioned hereditary tendencies, developmental pneumatization defects, special tissue sensitizations, allergies, vitamin deficiencies, dysendocrinias, malnutritions, metabolic disturbances, and secondary effects of other pathogenic agencies—fatigue, trauma, emotion, exposure, intoxication, or any combination of these and other noxious influences.

The subject matter presented ignores the fact, alluded to yesterday by Dr. Barnhill, that years of bacterial infestation of the individual's respiratory structures without onset of disease, have preceded ethmoiditis, yet when ethmoiditis does develop it is at once attributed to bacterial etiology. Bacteria take part in the pathogenesis, but rarely as prime factors. The various etiologic factors just alluded to which bring about bacterial tissue invasion are the prime etiologic factors of infectious tissue invasions; as such they are the particularly important things from the standpoint of diagnosis and treatment. In formulating therapeutic measures adapted to an individual case, etiology is the logical starting point. It seems to me that such thoughts might be added to the valuable contributions of this symposium with some advantage.

Dr. John A. Pratt, Minneapolis, Minn.: Dr. Mosher in his excellent paper has proven:

1. That the agger nasi cell is not a proper entrance to the ethmoid capsule because it is only present in one-third of the cases.

2. That the high-up mesial wall entrance is extremely dangerous to all except the most skilled operator, because a slip of the curette and the cribriform is entered and death ensues.

3. That the ethmoid capsule is a definite bony cavity filled with cells that block the entrance to the frontal sinus and can be exenterated without danger if the middle turbinate is left in situ.

4. That the anterior ethmoid cells must be exenterated for a safe entrance intranasally to the frontal sinus.

5. By Fig. No. 11, that the easiest and nearest approach to the ethmoid capsule is under the anterior attachment of the middle turbinate into the anterior ethmoid cells.

6. That wandering ethmoid cells, so often heralded in the past as contraindications for the intranasal operation, are only present in one out of twenty cases, so he gives us the opportunity to cure 95 per cent. of the ethmoid cases and leave 5 per cent. for the external operation.

All these conditions I advocated in my "Intranasal Operation for the Exenteration of the Ethmoid Capsule," presented to this society in Cleveland in 1919.

I cannot agree with Dr. Skillern when he states that the normal mucous membrane is dry. All mucous membrane must have functioning moisture; when above or below this amount it is pathological. Dr. Skillern seems willing to sacrifice anything in the nasal cavity except the ethmoid cells. I wish the Doctor would tell us what physiological benefit we derive from the ethmoid cells.

I have not had the beneficial results from suction that Dr. Iglauer reports. In fact, I have thrown a patient into an occlusion of the sinus ostia by passive congestion from suction. I prefer middle turbinate infraction followed by glycerine base medicinal packs for hygroscopic effect. As soon as possible the offending septum is operated upon, and then, if necessary, the ethmoid cells are exenterated.

With the proven fact that infection follows the cut sheaths of the olfactory nerves and causes meningitis (Present Status of the Ethmoid Operation, A. M. A., June, 1924, Chicago), the removal of the middle turbinate should have serious consideration. Believing, as we do, that thick or deflected septa are the causus belli of nasal disorders, they should have our first attention. In exenterating the ethmoid capsule there is no need to sacrifice that protector of the lateral wall, the middle turbinate. The operation can always be performed without its removal. My operation for reduction of the cystic middle turbinate (A. M. A., June, 1922, Boston), makes its removal unnecessary.

Dr. Fenton has very aptly stated that to do intranasal surgery one must have anatomical knowledge, orientation and surgical touch.

The technique for external operations of Dr. Lynch seems beyond criticism.

Dr. S. Fineman, New York City: I have a slide showing the pitfalls in x-ray of the ethmoids—three films of the same patient taken ten minutes apart. One film shows *left* cloudy ethmoids; the next shows *right* cloudy ethmoids, and the third shows both sides *clear*. The reason—usually not recognized by the rhinologist is faulty x-ray technique.

DR. BURT R. SHURLY, Detroit: This undoubtedly is one of the most interesting subjects that the otolaryngologist has to deal with, due to the fact that there is a wide difference of opinion, the pendulum swinging all the way from ultra-conservatism to ultra-radicalism. Throughout a period of twenty-five years we have had an enormous amount of literature, some splendid scientific work, and yet today we have all these various viewpoints, various methods and technique, carefully worked out and brought together in this symposium. It seems to me that those of us who practice in this line realize that the public has a knowledge of the subject so that today the individual who comes to our office comes with more or less of an order from his pediatrician, or his internal medicine consultant, for a sinus operation. He comes to us as sinus men; he comes to us with a new sort of education. Whereas formerly he had a cold in the head, now he has sinus disease.

If we are to orientate this subject, analyze and summarize it, it would seem to me that we must bear in mind certain definite scientific truths that are operative continuously. First of all, that our diagnostic procedure is carefully executed along certain definite scientific lines. This is our armamentarium and, together with those wonderful laws of common sense, surgical judgment, and a careful consideration of the individual case, we may decide what our procedure must be in this particular case, and while we have scientic views as to the etiology of sinus disease, through our research experiments we are thoroughly convinced that the cell in its resistance to the virulence of infection varies with the individual and with the various climates of the country. Where one individual will succomb, or develop an increased amount of pathology, another will have a modified pathology, according to the resistance of the individual, or the climate in which he lives. Consequently it is necessary to bear all these various manifestations carefully in mind, and the procedure can be a middle-of-the-road procedure for a large number of patients. The results can be wonderfully good according to the combined efforts that we use surgically and along every other line.

In our research work we find that with vitamin A deficiency three particular germs invade these sinuses, the resistance is lowered and a certain definite pathology develops. There may be a world of usefulness that will come out of more scientific experiments along this line, as we develop the facts of the resistance of the cell. In the meantime, we can very carefully follow the middle of the road in surgical procedure which will allow us individually to exercise our surgical judgment, with the special training that we have along surgical lines, very carefully watching for the danger points and very carefully conserving the natural conditions which we know so well exist in the nose.

DR. RALPH A. FENTON, closing: I have only to say for the benefit of my good friend, Dr. Lewis, that I would like to call his attention to the specific contraindications with which my paper is profuse. I did not say that all such cases have to be operated on. Furthermore, I know Gene operates—he is one of the best surgeons of my acquaintance.

It is important also to realize that Dr. Skillern's remarks in regard to pathology are with particular reference to the extreme case which de-

mands operative procedure. We all know that there is a tremendous number of cases of ethmoid disease which do not require operation; and of course the radical operation which is successful is very limited in its application to this field of ethmoidal disease.

DR. HARRIS P. MOSHER: I would like to ask Dr. Beck to answer a question that is asked of me by a man who has a chronic suppurative ethmoid and bronchiectasis. He asks, "What can you promise me by operating on my ethmoids in regard to the bronchiectasis?" I would like to have Dr. Beck answer that.

DR. JOSEPH C. BECK, Chicago, Ill.: That is a question entirely away from the subject of this symposium.

DR. MOSHER: The whole ethmoid question is now open for discussion.

DR. BECK: I do not know whether I could promise to cure him, because the question of the bronchiectasis is to be considered and not the ethmoid. If the bronchiectasis has existed a sufficient length of time that changes in the lung have occurred, no operation on the sinuses will cause recovery because it is already a lung condition. I cannot see how anyone could promise anything, but I will say that in many bronchiectasis we have had excellent results by operation on the sinuses, particularly when non-suppurative.

If I may be allowed to continue the discussion I would like to say that Dr. Lynch's paper is simply the starting point. He speaks of the external route, presumably through the face. There is an external route through the antrum. The operation which should not be discarded is the trans-antral ethmoid operation. Recently quite a number of excellent results have been reported.

INSTRUCTIONAL SUBJECTS

FRANCIS HEED ADLER, M. D., Philadelphia, Pa.

The Choice of Glaucoma Operation.

Indications for and Discussion of the Various Operations Used in Glaucoma.

CHAS. A. BAHN, M. D., New Orleans, La.

Glandular Therapy in Ophthalmology.

S. J. BEACH, M. D., Portland, Me.

Testing of Vision Including Malingering Test Charts, Proper Illumination, Notation of Acuity, Use of Astigmatic Charts, etc.

CONRAD BERENS, M. D., New York City

Physiology of the Ocular Muscles With Special Reference to Fatigue of Accommodation and Convergence.

F. J. BRIBACH, M. D., Atchison, Kan.

Management of Commoner Forms of Conjunctivitis.

W. H. CRISP, M. D., Denver, Colo.

Determination of Astigmatic Axis and with Cross Cylinder: Demonstration on Members of Class.

RICHARD GAMBLE, M. D., Chicago, Ill.

Histopathology of Disease of the Macular Retina.

Histopathological Demonstrations of Macular Diseases. (This should be supplemented by Dr. Bedell's Course No. 47.)

EVERETT L. GOAR, M. D., Houston, Tex.

Dracryostenosis and Dacryocystitis.

Diagnosis, Nonsurgical and Surgical Treatment.

F. HERBERT HAESSLER, M. D., Milwaukee, Wis.

Ocular Tuberculosis.

Diagnosis and Appearance with Slit Lamp, Ophthalmoscope, etc.

Differential Diagnosis, Treatment and Prognosis.

EDWARD JACKSON, M. D., Denver, Colo.

Ophthalmic Literature.

What to Read, How to Compile Bibliography, How to Write.

W. B. LANCASTER, M. D., Boston, Mass.

The Magnet.

Various Types of Magnets and Their Use.

S. HANFORD McKEE, M. D., Montreal, Canada

Bacteriology of the Conjunctiva.

The Commoner Organisms and Their Clinical Manifestations.

JAS. M. PATTON, M. D., Omaha, Neb.

Inter-relation of Ophthalmology and Otolaryngology.

Affections of the Eye Due to Otolaryngic Conditions and Vice Versa.

BERNARD SAMUELS, M. D., New York City

Viterous Opacities.

Cause, Recognition, Pathology and Treatment.

EDMUND B. SPAETH, M. D., Philadelphia, Pa.
The Correction of Lid Deformities.
Discussion of Various Operative Procedures.

E. STIEREN, M. D., Pittsburgh, Pa.
The Care of Recent Ocular Injuries.
The immediate Care of Eye Injuries and Prevention of Complications.

R. VON DER HEYDT, M. D., Chicago, Ill.
Photography of the Living Eye.
Stereophotography of the External and Anterior Eye,
Photography of the Fundus, Demonstration.

ARTHUR J. BEDELL, M. D., Albany, N. Y.
Clinical Aspects of Diseases of the Macular Retina.
Discussion and Demonstration of Actual Photographs of the Conditions.

F. J. BRIBACH, M. D., Atchison, Kan.
Management of Commoner Forms of Conjunctivitis.

A. B. BRUNER, M. D., Cleveland, Ohio
Use of Telescopic Aids to Vision.
Indications for and Fitting of Telescopic Spectacles and Magnifiers.

MARTIN COHEN, M. D., New York City
Tonometry.
Tonometers, Method of Use, Value of Results.

ALFRED COWAN, M. D., Philadelphia, Pa.
Cylinder Retinoscopy.
Actual Retinoscopy on Model Eyes with Explanation of Theory.

E. C. ELLETT, M. D., Memphis, Tenn.
Concerning Forms of Secondary Glaucoma.
Various Forms. Recognition and Treatment.

J. N. EVANS, M. D., Brooklyn, N. Y.
Central Field Scotometry.
Indication for and Methods of Central Field Scotometry.

A. GREENWOOD, M. D., Boston, Mass.
The Operative Treatment of Squint.
Various Operations Employed, Indications and Technic.

F. HERBERT HAESSLER, M. D., Milwaukee, Wis.
Ocular Tuberculosis.
Diagnosis and Appearance with Slit Lamp, Ophthalmoscope, etc.,
Differential Diagnosis, Treatment and Prognosis.

EMORY HILL, M. D., Richmond, Va.
Ophthalmic Therapeutics.

A. N. LEMOINE, M. D., Kansas City, Mo.
The Use of Tuberculin in Ophthalomolgy.
Indications For and Technic of Use of Various Tuberculins.

LUTHER C. PETER, M. D., Philadelphia, Pa.
Perimetry.
Methods, Interpretation of Results.

GEO. DE SCHWEINITZ, M. D., Philadelphia, Pa.
Vascular Diseases of the Fundus.
Significance of the Fundus Findings.

DORLAND SMITH, M. D., Bridgeport, Conn.
Refraction Without Mydriatic.
Methods and Advantages of Refraction Without Mydriatic.

DAVID W. WELLS, M. D., Boston, Mass.
Development of the Fusion Sense.
Consideration of the Fusion Sense.
Importance, Means of Development.

J. C. BECK, M. D., Chicago, Ill.
Histopathology of the Larynx.
Euscope Demonstration of Histopathology of the Commoner Diseases of the Larynx.

GEO. M. COATES, M. D., Philadelphia, Pa.
Conservative Surgery of Mastoid and Middle Ear.
Indications for Surgery.
Indications for Conservatism, Methods.

RALPH A. FENTON, M. D., Portland, Ore.
Vertigo. Discussion of Forms, Ocular and Aural.

S. J. KOPETZKY, M. D., New York City
Hematologic Pictures in Otology.
The Blood Pictures Presented in Various Diseases of the Ear and Their Significance.

W. I. LILLIE, M. D., Rochester, Minn.
Cerebellar Pontine Angle Tumors. Diagnosis.

EDWARD LODHOLZ, M. D., Philadelphia, Pa.
Physiology of the Nose.
The Physiology Processes that Occur in the Nose and the Physiological Reasons for the Anatomy of the Nose.

WM. J. McNALLY, Montreal, Canada
Physiology of the Labyrinth.

DOUGLAS MACFARLAN, M. D., Philadelphia, Pa.
Aids to the Hard of Hearing.
Demonstration of Various Instruments to Help Deficient Hearing

WM. V. MULLIN, Cleveland, Ohio
Diseases of Salivary Glands.
Diagnosis, Treatment.

FRANK NOVAK, M. D., Chicago, Ill.
Complications of the Common Cold.
Prevention, Diagnosis, Treatment.

LEROY M. POLVOGT, M. D., Baltimore, Md.
Otosclerosis.

ARTHUR W. PROETZ, M. D., St. Louis, Mo.
Roentgenographic Studies of Disease of the Paranasal Sinuses.
Methods of Visualization of Sinuses, Diagnostic Value.

JOHN J. SHEA, M. D., Memphis, Tenn.
Acute Sinusitis.
Methods of Diagnosis and Treatment.

R. SONNENSCHEIN, M. D., Chicago, Ill.
Hearing and Tuning Fork Tests.
Demonstration and Interpretation of the Various Tests Used.

SIDNEY YANKAUER, M. D., New York City
Surgery of the Septum Nasalis.
Indications for and Technic of Various Operations Upon the Septum.

A. H. ANDREWS, M. D., Chicago, Ill.
External Diseases of the Ear.

JOHN BARNHILL, M. D., Miami Beach, Fla.
Syphilis of the Respiratory Tract.
Syphilitic Affections of the Mouth, Nose, Pharynx, and Larynx.
Diagnosis, Prognosis, Local Treatment.

T. S. BLAKESLEY, Kansas City, Mo.
Physical Agencies in Otolaryngology.
Heat, Cold, Massage, Vibration, Ultra-violet, Infra-red, Diathermy, etc.

RALPH A. FENTON, M. D., Portland, Ore.
Vertigo.
Discussion of Forms, Ocular and Aural.

PERRY G. GOLDSMITH, M. D., Toronto, Canada
Local Therapeusis in Otolaryngology.
Indications and Applications of Therapeutic Means Other Than Surgery.

S. IGLAUER, M. D., Cincinnati, Ohio
Peritonsillar Abscess and Deep Cervical Abscess.

V. H. KAZANJIAN, M. D., Boston, Mass.
Plastic Surgery of the Nose.
Operative Procedures to Improve Nasal Appearance.

JOHN A. KOLMER, M. D., Philadelphia, Pa.
Perennial Rhinitis and Hayfever.
Causes, Clinical Manifestations, Differential Diagnosis, Treatment.

DOUGLAS MACFARLAN, M. D., Philadelphia, Pa.
Aids to the Hard of Hearing.
Demonstration of Various Instruments to Help Deficient Hearing.

JOHN MYERS, M. D., Kansas City, Mo.

Pain in Otolaryngology.

Causes, Pathology, Treatment.

HORACE NEWHART, M. D., Minneapolis, Minn.

The Audiometer.

Demonstration of Instrument, Use in Individual and Group Testing.

LEROY A. SCHALL, M. D., Boston, Mass.

Histopathology of Mucous Membrane of Nose and Nasal Sinuses.

ROSS HALL SKILLERN, M. D., Philadelphia, Pa.

Ethmoidal Disease. General Discussion.

JOHN J. THEOBALD, M. D., Chicago, Ill.

Routine Testing of Hearing of School Children.

Methods of Testing, Results Obtained in 28,000 Cases, Methods of Record.

PRESENTATION OF INSTRUMENTS AND APPLIANCES

A FUNDUS COLORIMETER

MARTIN COHEN, M.D.

NEW YORK, N. Y.

We have long felt the need of a device for recording the increasing or decreasing pallor of the optic disc and other fundus areas. This instrument I have devised to serve as a clinical aid in the diagnosis and prognosis of such conditions as retro-bulbar neuritis, multiple sclerosis, tabes, brain affections, accessory sinus diseases, slight color differences between the optic discs, and other pathological conditions. Our judgment of progressive and retrogressive changes in the color of the disc depends upon our visual impression of previous ophthalmoscopic examinations which at times are difficult or impossible to recall. We also infer disc color changes from examination of the visual acuity and perimetric tests; these latter tests are of subjective value and are frequently unreliable, owing to the patient's poor vision or lack of cooperation. Whether this instrument will detect color changes earlier than the functional tests cannot be stated, as clinical study in this respect has not yet been completed.

Two years ago I started to develop this instrument, and after careful consideration decided upon a colorimetric wedge principle to be applied in its development. I finally concluded to have constructed a calibrated magenta circular wedge-filter, to be attached to the electric hand ophthalmoscope.

The spectro-photometric principle involved in the construction of the circular wedge-filter was supervised by Dr. Jones at the Research Laboratory of the Eastman Kodak Company. It consists of gelatin stained with very red magenta; the wedge has a clear color near its apex, then takes on a faint reddish tint of relatively low saturation, which gradually becomes a deep red color towards its base. It is 18 m.m. in diameter, 3 m.m. in width, 2 m.m. thick, with a hole in the centre for mounting, and is sealed with Canada balsam between cover glasses. After the wedge was perfected, it was sent to Mr. Poser of Bausch & Lomb Optical Company, who had it attached with calibration to

the hand ophthalmoscope, the instrument thus serving as an electric ophthalmoscope and colorimeter.

A narrow, thin, graduated scale surrounds the circular red wedge, reading from zero at the apex of the wedge to 100 at its base, with division into ten equal parts. The circular wedge with its surrounding scale is attached to the ophthalmoscope and to a rod having at its lower end a wheel that is readily turned, thereby producing a revolution of the combined graduated scale and red wedge. The light from the electric bulb of the ophthalmoscope passes upward through a circular aperture in which the circular wedge and scale revolve. The light then comes in contact with the total reflecting prism of the ophthalmoscope, passes into the patient's eye, and is then reflected back into the examiner's eye.

The light which enters the patient's fundus through the clear apical portion of the wedge appears as a normal whitish disc, similar in size to the normal papilla. This whitish disc is used for fundus examinations. If the color changes in the optic

disc are to be studied, the colorimeter is brought into play by gently revolving the wheel which is conveniently located near the handle of the ophthalmoscope. This procedure very gradually changes the color of the whitish projected disc from low to high saturations of red. As soon as the whitish disc on the papilla commences to appear reddish, its reading,—which is readily seen on the instrument,—is noted; this notation is used for comparison in future examinations of the same case. In most normal cases the incipient color change to a reddish hue of the temporal side of the disc is noted at a reading of ten on the scale. Changes in scale reading at consecutive periodic examinations, if greater than ten, are suggestive of a progression or retrogression of the pathological lesion.

To conduct the colorimetric examination, it is necessary to dilate the pupils, to have the refraction properly corrected by lenses in the ophthalmoscope. The illumination from the ophthalmoscope should be of the same brightness at each subsequent colorimetric examination; this can be practically determined by observations of the illumination on ground glass; its intensity should be always maintained.

It is well known that the color of the disc and other areas is not always a criterion of pathology, but if color effects increase or decrease it is usually due to a pathological lesion.

The colorimetric fundus test is of value solely for individual patients, not in comparing results in different individuals. They should always be conducted by the same examiner, for the reason that color differences and color impressions vary in patients and examiner.

I am unable at present to give a detailed report on results of a sufficient number of cases with respect to the value of this instrument, but I have been encouraged to note its diagnostic aid in two cases of progressive disc pallor due to a unilateral chronic retro-bulbar neuritis, in which the visual acuity and perimetric tests could not be satisfactorily determined. In three cases of tabetic optic atrophy, I have observed an increase in the colorimetric measurement.

Further clinical study of the value of this instrument will be reported at a future meeting.

CROSS RETINOSCOPE

GEORGE H. CROSS, M. D.

CHESTER, PA.

DR. GEORGE H. CROSS, Chester, Pennsylvania: Finding that I was having trouble in seeing the edge of the pupil sharply with the ordinary retinoscope, I first resorted to the addition of a spherical + 1 lens held in back of the opening in the mirror, it however was a nuisance to handle so I then wore a pair of spectacles with a spherical + 1 added to my distant correction, this necessitated the constant changing of glasses. Knowing how the old Leibreicht ophthalmoscope was made to add different lenses back of the peep hole, it was thought to improve on it by using a spherical + 1 as used in a Loring ophthalmoscope and place it under the mirror in a well cut out of the metal base

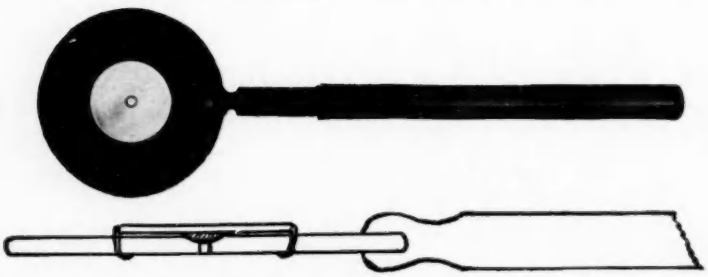


plate to which the mirror was attached. This was so satisfactory that I thought the idea should be passed on to those who would like to try it. It is light, handy, and unnecessary to change glasses, nor do you have to take into consideration any extra allowance for the one meter working distance. For those of us who are a little bit beyond the age when presbyopia begins, it will be a great help in sharpening the edge of the pupil so the shadow is much clearer. It makes retinoscopy much more of a pleasure than formerly due to the elimination of the clouding and difficulty in seeing the edge of the shadow. Messrs. McIntire, Magee and Brown Co. of Philadelphia very kindly did the mechanical work to construct the instrument.

A NEW EYE LOUPE

By Walter Stevenson, M.D., F.A.C.S.

QUINCY, ILLINOIS

Presented by Dr. Samuel G. Higgins, Milwaukee, Wisconsin

The instrument is a Berger Loupe, modified in size, attached by a rigid upright to the wearer's spectacles. This may be raised or lowered to suit the operator's convenience. The wearer's correction can be placed in the spectacle frame, and there is quite sufficient room on either side of the Loupe and below it to permit peripheral vision outside the limitations of the Loupe area. Its advantages over the Beebe Loupe are: first, the larger field of vision through the Loupe; second, the Loupe is covered with an opaque hood, which permits better concentration of the visual rays. Attached to this Loupe is also a modified Spec Lite for illumination.

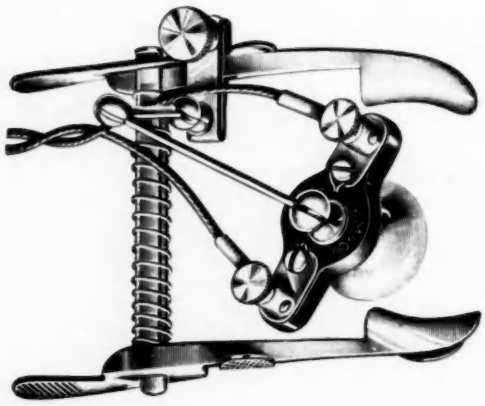
AN ELECTRIC CORNEAL PASTEURIZER

By Walter Stevenson, M.D., F.A.C.S.

QUINCY, ILLINOIS

Presented by Dr. Samuel G. Higgins, Milwaukee, Wisconsin

The second instrument was devised to take the place of the pasteurizer devised by Prince, of Springfield, Illinois. This, as you know, consisted of a copper ball, which it heated in an alcohol flame then held as close to the cornea as possible. The principle being, that the infra-red heat rays destroy the pneumococcus in serpiginous ulcerations. There can be no question about the value of this method of treatment, but there is always the danger that the operator's hand may slip, or that the patient may move, causing a severe burn of either the cornea, or the lids. Attached to a rigid eye-speculum, I have placed a small platinum electrode enclosed in a parabolic reflector. This can be placed at any distance from the corneal ulcer, and heat is produced by passing the ordinary city current through a reducer. I have had excellent results in several cases of corneal ulcerations with the use of this instrument.



A PERIMETER LIGHTING UNIT ATTACHED TO THE PATIENT'S HEAD

SIDNEY L. OLSHO, M. D.

PHILADELPHIA, PA.

The artificial illumination of perimeters has engaged very considerable attention for a number of years.

The problem is baffling because of the difficulty of securing controlled uniform, shadowless illumination of desirable color and candle power equal at all points on the movable arc, with no light shining in the patient's eye.

Inasmuch as the use of reflectors on all the devices now in use do not meet exacting requirements I propose an entirely new avenue of approach, not perfect, but having certain advantages which will become immediately apparent.

When we make a visual field the eye which is being examined naturally must be placed at the center of rotation of the perimeter arc. Obviously the light can not occupy that precise position.

I propose to use a laryngological head band with an attachment to hold a very concentrated light source not on the instrument but on the head of the patient. The light is placed just above the eye under examination, as near to that strategical center of rotation of the perimeter arc as possible. It may be shaded slightly below from the patient's eye or placed just beyond the line of vision. No reflector is employed.

The perimeter lighting unit shown today is not in the permanent form. The idea is there. The details will be perfected. As it now stands we have a socket holder, on a laryngological head band adjustable to a position above the eye to be examined. The socket carries a 32 C.P. 6-8 volt, Daylight bulb as used in an automobile headlight. The light filament in this lamp is as nearly round as I have as yet been able to obtain. We can not use a long coil as it would, except in one position, intersect the plane of the perimeter arm. With a perfectly round source of illumination placed where the eye is, the perimeter arm would

be ideally illuminated. In the position in which I place my lamp a very close approach is made to practical accuracy. Shadows from the brow and nose fall well to the outside of the normal field limits. If the holder is metal it may get too warm. Other material will be found. There has not been time to analyze the quality of the light. It is of sufficient intensity and the intensity is under control of a rheostat. The candle power illumination will be standardized as soon as practicable. Fields can then be made at any of several degrees of illumination.

Attached to the central knob of the head band is a hard rubber round eye-cover or disc which can be made to swing over the other eye and hold in place a piece of sterile gauze or cotton.

This illuminating unit can also be used with a 110 C.P. intermediate base, daylight sign-lamp without a rheostat. The unit can be used to advantage with the hand campimeter. It can be used for campimetry on a bed patient who is in the recumbent posture. The outfit is adaptable to any perimeter and the cost is extremely low.

RIVERBANK METALLIC RESONATOR

ROBERT SONNENCHEIN, M. D.

CHICAGO, ILL.

DR. ROBERT SONNENCHEIN, Chicago: I wish to present the Riverbank Metallic Resonator, the advantages of which are:

1. Durability.

2. Purity of tone.

3. Constant output at a given resonator setting. Practically no sound is transmitted through the supports of the resonator to the table. This is not true in the case of the ordinary wooden resonator.

4. Range of intensities made possible by tuning the resonator. The intensity may be varied by changing the area of the orifice of the resonator from zero to its maximum, at which point the resonator and fork are in tune. If a striking mechanism is used the setting of the resonator can be calibrated in terms of the intensity of sound output.

HEAD LAMP

T. S. Blakesley, M. D.

KANSAS CITY, MO.

Dr. T. S. Blakesley, Kansas City, Mo.: I wish to present a new head lamp that I have been working on for a number of years, and which has quite a number of advantages. The first one is that the mirror has a hole which is concentric; the mirror is rotatable and can be used on either side of the head. The

advantage of that is that one can get the mirror close to the nose, and it is possible to see through the hole and at the same time take the pupillary distance into consideration. The lamp can be thrown into focus for either throat with a large field, or a small field for laryngeal and bronchoscopic work. It throws parallel rays that will penetrate down a tube. After the lamp is on the head and one looks through the hole and gets the field one wishes to illuminate, it can be swung from side to side to get the focus you wish. It gives a brilliant illumination, simply wearing the mirror on the forehead and directing the light.

PILLAR SEPARATOR

W. D. Black, M. D.

ST. LOUIS, MO.

Dr. W. D. Black, St. Louis, Mo.: The advantages of this pillar separator are that after the tonsil is removed the instrument is quickly placed between the pillars and by pressing down the ratchet and pulling backward the finger guard the pillars are spread to any width you may desire, thus giving an excellent view of the tonsillar fossa. A small sponge may be necessary to wipe away the blood, but by the tension of the jaws or separators the bleeding becomes much less. It has a detachable handle and can be left in position if one so desires. The advantages are:

First: One is able to inspect the fossæ carefully.

Second: One can readily see where the bleeding vessels are and can apply hemostats with more accuracy.

Third: The tension on the pillars has a tendency to throw the tonsil bed inward towards the mouth and in so doing the arteries are contracted, causing less bleeding.

AN ACCOMMODATION ERGOGRAPH

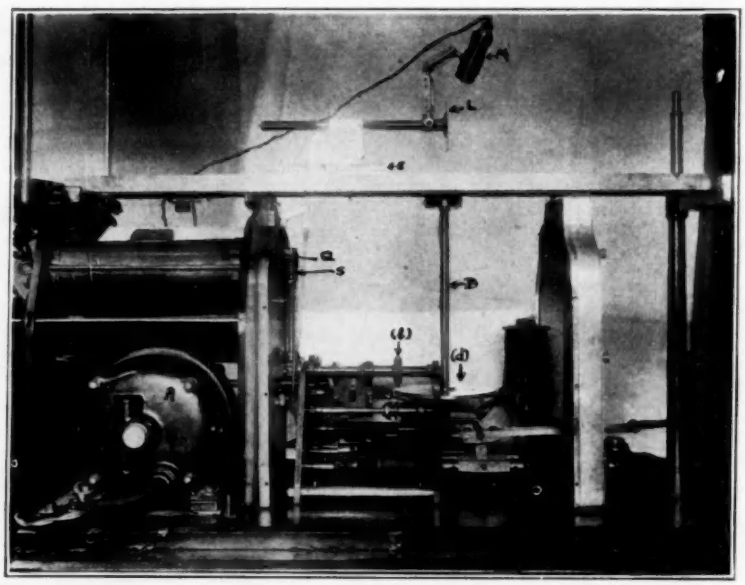
CONRAD BERENS, M.D.

NEW YORK, N. Y.

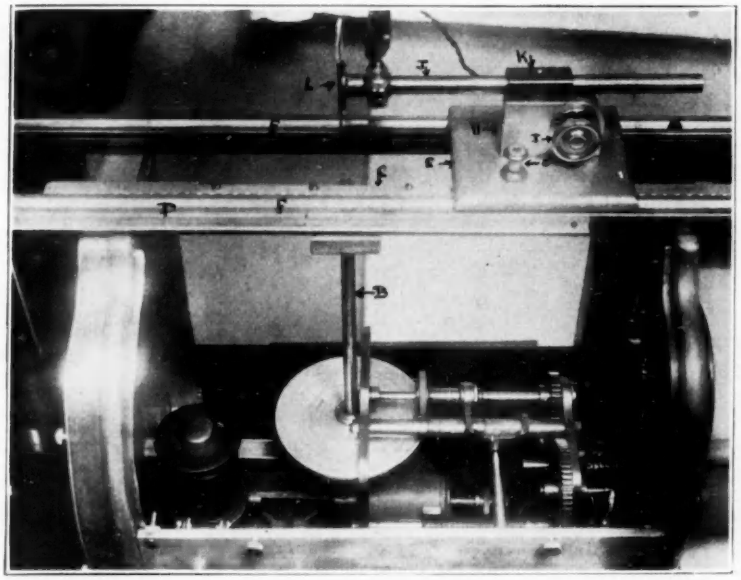
Lucien Howe[1] in 1916 described the first accommodation ergograph. The seventh accommodation-convergence ergograph† to be set forth here, was evolved from his model, and its construction, as well as that of the one previously described,[2] is in no small measure attributable to the encouragement of Doctor Howe and Doctor Wilmer.

DESCRIPTION OF ACCOMMODATION ERGOGRAPH, NUMBER SEVEN

This instrument was designed to overcome defects of previous models and has the advantage of automatically controlling the rest period, the rate of approach of the test object and its return to the base line position. It is driven by a General Electric 1/20 horse power, 110 volt, direct current motor (A, Fig. 1)



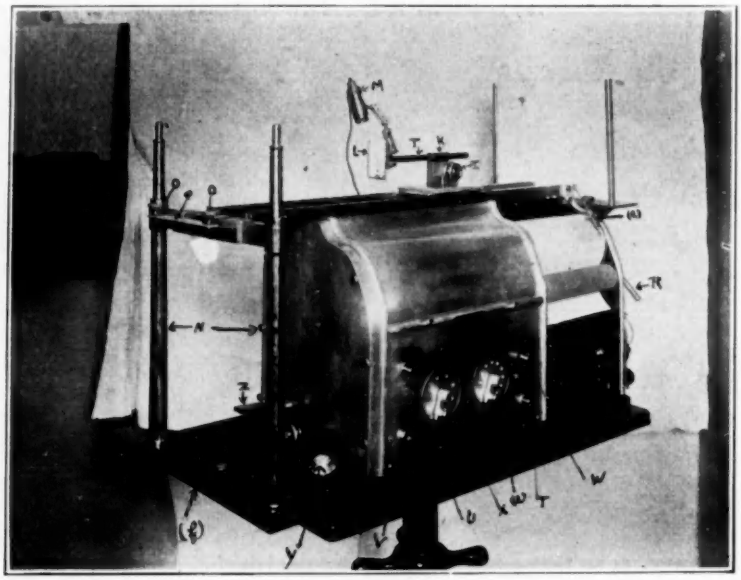
†Constructed by Stephen M. Balzer.



model 3 (Type SD, Form D) in circuit with a Ward Leonard 110 volt rheostat. All calculations are based on a speed of 1725 revolutions per minute.

The shaft from the motor drives an upright rod (B, Figs. 1 and 2) activating a flat rod (C, Fig. 2) 16¾ by 1 by ¼". This rod is placed between the tracks (D, D, Fig. 2) on which the test object carriage (E, Figs. 1 and 2) operates. Teeth milled for a space of 6¾ inches allow the test object to move six inches at one setting.

The carriage (E, Figs. 1 and 2) 3¾ by 3¾ by ½" is fitted into two V-shaped slots in two rods (F, F, Fig. 2) 24 by ¾ by ¾". This carriage moves along the entire length of the two supporting tracks and may be attached to the activating rod at any point by a small pin (G, Fig. 2) which fits into 3/16 inch holes drilled 1/16 inch apart in the flat activating rod. On the carriage is an upright support (H, Fig. 2) 3 by ½ by 1½" and set into this is a rod 3¾ by 1" milled on one side, so that it may be elevated by a thumb screw (I, Figs. 2 and 3). On this support a rod (K, Figs. 2 and 3) 1¼ by ½ by ½" pierced by a ⅜ inch hole receives a brass rod (J, Figs. 2 and 3) 7 inches long, at one end of which is a rectangular test object



holder (L, Figs. 1, 2, 3) 2 by 1½". The rod is hollow so that a light may be placed in it and the test objects illuminated from the rear.

A Leitz light (M, Figs. 1 and 3) fitted with focussing lens and daylight screen in circuit with a Leitz 110-220 volt rheostat is attached to this test object arm. The light is arranged above the test object to supply 7 foot candles of illumination.

Two uprights, (N, Fig. 3) 16 inches high, screwed into the frame at one end, are joined by a one inch cross piece 12 inches from the base. Two ½ inch projecting rods, 2½ inches long, are slotted to accommodate a wooden tongue depressor (P, Fig. 3) firmly held by two thumb screws (O, O, Fig. 3).

A cylindrical brass box (Q, Fig. 1) 6½ by 2" contains thin white tracing paper‡ used for recording. This passes over a brass drum 6½ by 3½". The paper is held in apposition and made to move with the drum by pressure of a rubber covered brass roller, 6½ by ½", on an eccentric. Pressure is applied by a lever (R, Fig. 3) and adjusted by a spring.

The large drum is geared to the motor by gear (S, Fig. 1).

‡Obtained from Eugene Dietzgen Co., New York.

The instrument board shows the motor switch (T, Fig. 3) which operates the motor, the accommodation switch (U, Fig. 3) which sets the test object carrier in motion. An electric plug hole (V, Fig. 3) receives a long cord for use in working at a distance. The lever (W, Fig. 3) marked K.Y.M. places the kymograph in neutral, high, or low speed. Lever (X, Fig. 3) marked REST PR SEC automatically holds the test object carrier at the distal end of the instrument for ½ to 7 seconds. This is accomplished by causing a governor to travel a shorter or longer distance on a threaded rod before it is released. The forward progress of the carriage may be stopped by pressure on key (Y, Fig. 3) conveniently placed at the subject's hand.

The carriage may be controlled entirely by hand by means of lever (Z, Fig. 3) which trips the solonoids, making pressure on the disc from above to drive the mechanism forward and from below to drive it backward.

The arm on the dial marked SPEED PR SEC. (a, Fig. 3) changes the forward speed of the carriage from 2 to 4 cm per second by moving rubber disc (b, Fig. 1) nearer to or farther from the center of the metal disc attached to the upright which activates the carriage. The speed of return of the test object may be varied by setting the rubber disc (c, Fig. 1) nearer to or farther from center of metal disc (d, Fig. 1). Recording is done by means of recording pen (e, Fig. 3) and red ink.§

The whole is attached to a solid cast iron base (f, Fig. 3) 26 by 12 by ¾". Three ⅜" holes drilled in the base permit its being bolted to a table or to a box for shipping.

Method of Conducting the Test

Preliminary

A complete medical history is taken. When necessary a physical examination and laboratory studies are also made. Routine examination of the eye includes refraction under a cycloplegic and study of the ocular muscles[3].

At the time of the test the following data should be recorded:

Name, date, diagnosis. Type of test, example, sustained accommodation R & L eye. Lenses worn. Punctum proximum

§Pen and special ink obtained from Leeds and Northrup, Philadelphia.

and punctum remotum of accommodation, each eye and both eyes. Near point of convergence in millimeters from the cornea. Muscle balance at 6 M and 25 cm. with Maddox rod and screen.

Technic of Conducting the Repeated and Sustained Effort Tests

1. The Repeated Effort Test.

The instrument is placed on an adjustable perimeter table and the height regulated until the patient can bite firmly and comfortably upon the wooden tongue depressor (P, Fig. 3) attached to the ergograph. Depending upon whether the right, left, or both eyes are to be used, the patient bites the tongue depressor on the right side, the left side, or in the center. By turning the thumb screw on the carriage the vertical position of the test object is adjusted so that it is on a level with the eyes. Uniform illumination of the test object is secured by regulation of the light on the carriage. The rod activating the test object carriage is placed at the end of the tracks, the carriage is separated from the rod by withdrawing the pin which fastens it, and the test object then brought to the near point of accommodation ten times. The average near point is computed in millimeters.

The carriage is now pinned to the activating rod 20 mm. farther away from the patient than the average near point of accommodation, allowing 20 mm. excursion. It was found that with longer excursions it was extremely difficult to fatigue accommodation, except in patients whose accommodation was very weak. In some instances it is necessary to lengthen the excursions to 30 or 40 mm., and after a few seconds to 60 or 80 mm. The test object carriage is then set in motion by pulling out the motor pin on the instrument board. The subject is requested to press the button which makes the test object return to its base position when the test letters become actually blurred. For a test object several letters subtending a visual angle of 5 minutes at 250 or 300 mm. are used. Since macular fatigue occurs very rapidly, the patient is instructed to shift fixation quickly from letter to letter. If the Duane line is used the patient is instructed to shift fixation from the top to the bottom of the black line. The rest period, which may be adjusted from one half to seven seconds, is arranged for one half second's rest by adjusting the lever on the instrument dial. In this way the test object remains at the base position only long enough to be

driven forward again. Unless the rest periods are very short, the mechanism of accommodation is so rested between efforts that it is impossible to produce fatigue even in patients with subnormal accommodation.

The speed of movement of the carriage is usually adjusted by means of the lever so that the test object moves at the rate of 4 cm. per second, both in approaching and receding from the near point of accommodation. It is particularly important that the test object be receded as rapidly as possible, to the far point allowed, for unless this is done the rest period is greatly lengthened. Provided the subject brings the test object to the near point each time, it is theoretically possible with this apparatus to make 40 to 45 efforts of accommodation per minute. Even in the repeated effort test, the ciliary muscle is continuously on tension so that complete rest of the mechanism of accommodation is not permitted. The kymograph is usually set at slow speed so that the records will occupy a small space in the files.

2. The Sustained Effort Test.

When it is impossible, because of marked fluctuation of the near point, to use the repeated effort test, it is advisable to use the sustained effort test. The preliminary procedure is the same as in the repeated effort test. The average near point of accommodation is determined, and with the test object at the base position, 20 mm. excursion is allowed. The subject then attempts to hold the test object as close to the near point of accommodation as possible; this means that very short repeated accommodative efforts must be made. This is done by using the lever which controls the forward and backward motion of the test object, and it is advisable to have the test object approach and recede from the near point at a speed of 2 cm. per second.

In attempting to evaluate the records of repeated effort and sustained effort tests, proper consideration should be given to the many variable factors involved, and hasty conclusions should not be drawn from one test.

Comment

It has been pointed out[4] that of 195 records made with the two previous models, only 30% showed any evidence of fatigue as indicated by recession of the near point of accommodation. Of the subjects who developed recession of the near point during

the test, 63% showed such rapid onset of fatigue as to require them to discontinue the test before the end of the 10 or 15 minute run.

We do not think that all patients whose accommodation is normal according to the Duane standard can be excluded, without a fatigue test, from the group suffering from accommodative asthenopia since the correlation between the Duane standard for age and ability to complete a 10 to 15 minute ergograph test is not higher than +48.

We believe the ergograph has furnished us a medium for graphically recording the behavior of the near points of accommodation and convergence which has aided in the differential diagnosis of asthenopia. It has afforded a means of observing progress and has stimulated the interest and cooperation of patients in general medical and orthoptic treatment.

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MINUTES.

American Academy of Ophthalmology and Otolaryngology

1929

Tuesday Morning *Joint Session*

The thirty-fourth Annual Meeting of the American Academy of Ophthalmology and Otolaryngology was held at the Traymore Hotel, Atlantic City, October 21st to 25th, 1929.

The Tuesday morning session was called to order at nine o'clock, the president, Dr. Harris P. Mosher of Boston, presiding.

The President: Gentlemen of the Academy and Guests: By virtue of the authority vested in me as President of this Academy, I now declare the thirty-fourth annual session of the Academy to be open for the regular transaction of business and for the scientific program.

Dr. Charlton is evidently busy for the moment. He will be here later with any announcements he wishes to make.

Dr. Harris P. Mosher, Boston, read the president's address entitled "The Submaxillary Fossa, Approach to Deep Pus in the Neck," First Vice-President Dr. Frank E. Burch, presiding.

Dr. Coulter Charlton of Atlantic City, local chairman of the Committee on Arrangements, reiterated the various announcements contained in the program.

The President introduced the Honor Guest, Mr. Herbert Tilley, London, England, who read a paper entitled "Some Experiences in the Surgical Treatment of Inflammation of the Frontal Sinuses and the Possible Complications."

SYMPOSIUM ON BRAIN ABSCESS:

The following papers were presented in this symposium:

From the Standpoint of the Ophthalmologist,
Dr. W. L. Benedict, Rochester, Minn.

From the Standpoint of the Otolaryngologist,
Dr. Wells P. Eagleton, Newark, N. J.

From the Standpoint of the Brain Surgeon,
Dr. William J. Mixter, Boston

Methods of Drainage,
Dr. Harry P. Cahill, Boston

This symposium was discussed by Drs. J. J. King, New York City, and S. J. Kopetzky, New York City.

SECTION ON OPHTHALMOLOGY

Tuesday Afternoon Program

The Tuesday afternoon session was called to order at two-thirty, First Vice-President Frank E. Burch, presiding.

Dr. William D. Rowland, Boston, read a paper entitled "Visual Field Studies: Marked Concentric Contraction of the Visual Fields. A Study of 100 Consecutive Cases." This paper was discussed by Drs. Luther C. Peter, Philadelphia; Mark J. Gottleib, New York City; Howard McI. Morton, Minneapolis; and William D. Rowland.

Dr. Georgiana Dvorak Theobald, Chicago, read a paper entitled "The Frequency of Sympathetic Ophthalmia." This paper was discussed by Drs. W. C. Camp, Minneapolis; Allen Greenwood, Boston; and Georgiana Dvorak Theobald.

Dr. F. N. Knapp, Duluth, Minnesota, read a paper entitled "Conservation of the Lacrimal Sac." This paper was discussed by Drs. E. C. Ellett, Memphis, Tennessee; John A. Pratt, Minneapolis; Ray K. Daily, Houston, Texas; A. S. Green, San Francisco; and F. N. Knapp.

Dr. Joseph L. McCool, San Francisco, read a paper entitled "Some Original Experiments with the O'Connor Muscle Shortening Operation." This paper was discussed by Drs. George F. Suker, Chicago; Allen Greenwood, Boston; and Joseph L. McCool.

Dr. George H. Cross, Chester, Pennsylvania, read a paper entitled "Removal of a Number Eight Lead Shot from the Vitreous with 20/20 or 6/6 Vision. Case Report." This paper was discussed by Drs. Allen Greenwood, Boston; E. L. Jones, Cumberland, Maryland, and George H. Cross.

The Tuesday afternoon session adjourned.

SECTION ON OTOLARYNGOLOGY

Wednesday Afternoon Program

The Wednesday afternoon session convened at two o'clock, the President, Dr. Harris P. Mosher, presiding.

Dr. W. J. McNally, Montreal, Canada, read the Report of Research Fellow in Otolaryngology, entitled "An Analysis of the Limb Responses to Semicircular Canal Stimulation in the Frog." There was no discussion of this report.

Dr. Robert Sonnenschein: I have permission at this time to present to you an instrument which has come to your Committee on Standardization of Tuning Forks and Hearing Tests. As you all know, resonators are instruments for amplifying sound. The advantages of this Riverbank Metallic Resonator are:

1. Durability.
2. Purity of tone.
3. Constant output at a given resonator setting. Practically no sound is transmitted through the supports of the resonator to the table. This is not true in the case of the ordinary wooden resonator.
4. Range of intensities made possible by tuning the resonator. The intensity may be varied by changing the area of the orifice of the resonator from zero to its maximum, at which point the resonator and fork are in tune. If a striking mechanism is used the setting of the resonator can be calibrated in terms of the intensity of sound output.

Dr. John F. Barnhill, Indianapolis, Indiana, read a paper entitled "The Relation of the Ear, Nose and Throat to General Infectious Disease." This paper was discussed by Drs. T. E. Carmody, Denver, Colorado; Judson Daland, Philadelphia; Eugene R. Lewis, Los Angeles, California; Lyman Richards, Boston; R. H. T. Mann, Texarkana, Texas; and John F. Barnhill.

Dr. Charles E. Connor, St. Paul, Minnesota, read a paper entitled "Laboratory Aids in Ear, Nose and Throat Conditions." This paper was discussed by Drs. Frank Spencer, Boulder, Colorado; Gordon Berry, Worcester, Massachusetts; Ralph A. Fenton, Portland, Oregon; S. J. Kopetzky, New York City; and Charles E. Connor.

Dr. Eugene R. Lewis, Los Angeles, California, read a paper entitled "Considerations Underlying Treatment of Sinus Disease." This paper was discussed by Drs. W. V. Mullin, Cleveland, Ohio; and Arthur W. Proetz, St. Louis.

Dr. Perry G. Goldsmith (and Dr. W. G. McGregor), Toronto, Canada, presented a paper entitled "A Consideration of Diseases of the Blood and Lymphatic Glands in Otolaryngology." This paper was discussed by Drs. Joseph C. Beck Chicago; Mark J. Gottleib, New York City.

Dr. Secord H. Large, Cleveland, Ohio, read a "Report: Foreign Clinics."

The Wednesday afternoon session adjourned.

Banquet.

The annual banquet was held in the Ball Room of the Traymore Hotel, Wednesday evening, October 23rd, at seven o'clock, the President, Dr. Harris P. Mosher, presiding.

The president introduced the President-Elect, Dr. William H. Wilder of Chicago; also the guest of honor, Mr. Herbert Tilley of London. After short responses, the President presented Dr. George H. Parker, Professor of Zoology, Harvard University, who made the address of the evening, entitled "Heredity."

Music was furnished by Lena Bricker, contralto; Charles Stahl, tenor, and Henry Lukens, accompanist, and the Hotel Traymore orchestra.

Thursday Afternoon Program

The Thursday afternoon session convened at two-fifteen, First Vice-President, Dr. Frank E. Burch, presiding.

Exhibition of Instruments

Dr. Martin Cohen, New York City, presented a fundus colorimeter.

Dr. Samuel G. Higgins, Milwaukee, Wisconsin, presented two instruments, a Berger loupe, devised by Dr. Walter Stevenson of Quincy, Illinois; also an instrument devised to take the place of the pasteurizer devised by Prince, of Springfield, Illinois.

Dr. Sidney Olsho, Philadelphia, presented a perimeter lighting unit that attaches to the patient's head.

Dr. George H. Cross, Chester, Pennsylvania, presented an improved retinoscope.

Dr. T. S. Blakesley, Kansas City, Missouri, presented an improved head lamp.

Dr. Conrad Berens, New York City, presented an accommodation ergograph.

SECTION ON OPHTHALMOLOGY

Dr. John M. Wheeler, New York City, read a paper entitled "Destructive Purulent Ophthalmia Accompanying an Eruptive Fever with Stomatitis. Case Report." This paper was discussed by Dr. W. C. Rutherford, Iowa City, Iowa.

Dr. Charles A. Young, Roanoke, Virginia, read a paper entitled "Recurrent Hemorrhages into Retina and Vitreous—Calcium Deficiency as Possible Cause." This paper was discussed by Drs. William Zentmayer, Philadelphia; William H. Crisp, Denver; and Charles A. Young.

Dr. Francis Heed Adler, Philadelphia, read a paper entitled "Reciprocal Innervation of the Extraocular Muscles." This paper was discussed by Drs. Walter B. Lancaster, and Francis Heed Adler.

Dr. Alfred Cowan, Philadelphia, read a paper entitled "Accessory Effects of the Correcting Glass by its Position before the Ametropic Eye." This paper was discussed by Drs. W. E. Shahan, St. Louis; Edward Jackson, Denver; Sidney A. Olsho, Philadelphia; E. E. Blaauw, Buffalo, New York; and Alfred Cowan.

Dr. F. Herbert Haessler (and Dr. Nelson M. Black), Milwaukee, Wisconsin, presented a paper entitled "Post Traumatic Ocular Tuberculosis." This paper was discussed by Drs. A. B. Bruner, Cleveland; Edward Jackson, Denver, Colorado; A. N. Lemoine, Kansas City, Missouri; and F. Herbert Haessler.

The Thursday afternoon session adjourned.

Business Session

October 24, 1929

The annual Business Meeting of the American Academy of Ophthalmology and Otolaryngology convened in the Ball Room of the Traymore Hotel, Atlantic City, at seven-thirty, Thursday evening, October 24, 1929, Dr. Harris P. Mosher, President of the Academy, presiding.

On motion of Dr. Burt Shurly, duly seconded, the reading of the minutes of the previous meeting was dispensed with.

The following reports were then presented:

Report of Secretary

1929

The Academy membership for the year just closed is as follows:

Reported at St. Louis meeting		1,476
Elected at the St. Louis convention		143
Total last year		1,619
For the present year:		
Dropped for non-payment of dues	2	
Deaths	25	
Resignations	9	37
Total		1,582
Divided as follows:		
Honorary Members	12	
Life Members	11	
Carried by Order of Council	4	
In good standing	1512	
One-year delinquent	27	
Two-year delinquent	16	1,582
Applicants pending:		
Complete	107	
Incomplete	56	163
Total carried on books		1,745

Death has claimed twenty-six, as follows:

Dr. J. M. Ball
Col. J. M. Banister
Dr. R. V. Brawley
Dr. E. B. Cayce
Dr. B. M. Dickinson
Dr. J. A. Ellegood
Dr. A. E. Ewing
Dr. C. H. Fellows
Dr. C. L. Frey
Dr. E. F. Gavin
Dr. Otto Glogau
Dr. F. B. Harding
Dr. T. C. Hood
Dr. Lucien Howe
Dr. L. W. Jessaman
Dr. D. M. Keith

Dr. J. S. Kirkendall
Dr. Lafayette Page
Dr. C. S. Rebuck
Dr. Charles W. Richardson
Dr. M. P. Rindlaub
Dr. E. L. Russell
Dr. William Hardin Sears
Dr. Greenfield Sluder
Dr. Myles Standish
Dr. Lewis H. Taylor

The following nine have resigned:

Dr. Carl Barch
Dr. E. M. Blake
Dr. J. E. Jennings
Dr. P. D. Kerrison
Dr. E. G. Linn
Dr. G. L. Noyes
Dr. H. M. Thompson
Dr. Will Walter
Dr. J. W. Wright

Two have been dropped for non-payment of dues:

Dr. Z. N. Short
Dr. E. S. Weimer

It has cost $18,249.61 to operate the society during the past fiscal year, or approximately $11.40 per member. The Pre-session Volume in 1928 cost approximately $1,700.00. So far as we can estimate, the cost this year will be about $1,300.00.

There are a large number of Transactions of former meetings on hand available to members wishing to complete their sets.

The sincere and active cooperation of all the executive officers is appreciated, and has made the coordinating work of the Executive Secretary and his office force function smoothly.

Respectfully submitted,

W. P. Wherry,
Executive Secretary.

On motion of Dr. A. J. Bedell, duly seconded, this report was adopted, and a vote of thanks extended to the Secretary.

REPORT OF TREASURER

October 5, 1929.

The American Academy of Ophthalmology and Otolaryngology, Cleveland, Ohio.

Dear Sirs:

In accordance with instructions received, we have examined the accounts of your Academy for the period of August 1, 1928, to August 1, 1929, and submit herewith a summary of receipts and disbursements for the period, together with a statement of the assets at August 1, 1929. We also attach a schedule of bonds owned at that date.

All receipts and disbursements were traced to the Academy's bank account, and the balance in the bank at August 1, 1929, was verified by a certificate from the depository. The cash balance at August 1, 1929, is made up as follows:

Deduct Vouchers not charged by bank:

Cheque in transit from Secretary (deposited August 16, 1929)		$308.77
Cash in bank (as shown by Bank Certificate)	$6,547.41	

Deduct vouchers not charged by bank:

DATE	PAYABLE TO	AMOUNT
Mar. 18, 1929	Leslie Patton, London, England	$100.00
July 30, 1929	Dr. W. P. Wherry	424.49
July 31, 1929	Dr. W. P. Wherry	264.34

Total vouchers outstanding	788.84	
Balance on deposit		5,758.57
Cash balance August 1, 1929		$ 6,067.34

Bonds on hand at August 1, 1929, were counted by us.

The following is a summary of the changes in the financial status of the Academy during the year.

Assets held August 1, 1928	$79,376.36

Excess of receipts on income over expenses:		
Receipts	$27,322.79	
Expenses	19,931.56	7,391.23
Assets August 1, 1929, per attached statement		$86,767.59

Yours very truly,

(Signed) PRICE, WATERHOUSE & Co.

SUMMARY OF RECEIPTS AND DISBURSEMENTS, AUGUST 1, 1928, TO AUGUST 1, 1929.

Cash in bank August 1, 1928		$ 4,505.61
RECEIPTS:		
Dues from members and application fees	$17,500.00	
Income from exhibitors	3,986.25	
Transactions	30.00	
Convention credit—net	838.00	
Interest on bonds, bank balances, etc.	4,513.54	
Profits on bonds sold	515.00	
	$27,382.79	
On Capital Account:		
Bonds sold—cost	12,775.00	
Total receipts		$40,157.79
		$44,663.40
DISBURSEMENTS:		
On Expense Account:		
Office and general expense	$ 5,629.30	
Expense of meetings	2,587.91	
Research work	4,500.00	
Transactions	5,833.04	
Honorariums	1,000.00	
Refunds	60.00	
Collections and exchange	52.47	
Accrued interest on bonds purchased	326.34	
	$19,001.56	

On Capital Account:		
Bonds purchased	18,604.50	$38,596.06
Cash in bank and on hand Aug. 1, 1929		$ 6,067.34

SUMMARY OF ASSETS AUGUST 1, 1929

Cash in bank and on hand	$ 6,067.34
Bonds owned—per attached list	80,700.25
	$86,767.59

SCHEDULE OF BONDS OWNED AUGUST 1, 1929

DESCRIPTION	INTEREST RATE	DUE	PAR VALUE	COST
Kansas City, Fort Scott & Memphis Railway Refunding Mortgage	4	1936	$5,000.00	$4,352.50
Chicago, Rock Island & Pacific Railway First and Refunding Gold Bonds	4	1934	6,000.00	5,337.00
Midvale Steel and Ordnance Company Convertible Sinking Fund	5	1936	17,000.00	16,669.00
Swift & Company Sinking Fund Gold Notes	5	1932	3,000.00	2,907.00
American Telephone & Telegraph Co. Sinking Fund Gold Notes	5½	1943	4,000.00	4,026.00
Denver Gas & Electric Company General Mortgage	5	1949	4,000.00	3,951.00
Witherbee Sherman Company First Mortgage Sinking Fund Series "A"	6	1944	5,000.00	4,925.00
Commonwealth Edison First Mortgage and Collateral Trust Series "C"	4½	1956	5,000.00	4,825.00
Detroit City Gas Company First Mortgage Gold Bonds Series "B"	5	1950	5,000.00	5,025.00

Kingdom of Denmark Gold Bonds	5½	1955	4,000.00	4,040.00
Youngstown Sheet and Tube Company First Mortgage	5	1978	5,000.00	5,003.75
Columbia Gas and Electric Company Debenture Bonds	5	1952	5,000.00	4,997.50
Van Sweringen Company First Mortgage Sinking Fund Gold Bonds	6	1952	9,000.00	9,047.00
Missouri Pacific First and Refunding Mortgages	5	1978	2,000.00	1,934.00
Chicago, Missouri and St. Paul and Pacific First Mortgage Gold Bonds Series "A"	5	1975	2,000.00	1,821.00
Goodyear Tire and Rubber Company First Mortgage and Collateral Trust Bonds	5	1957	2,000.00	1,839.00
			$83,000.00	$80,700.25

There being no objection, this report was received and filed.

Reports of Editor of the Transactions

To the Members of the American Academy of Ophthalmology and Otolaryngology.

Gentlemen: At the 1928 meeting of the Academy at St. Louis you were kind enough to re-elect me as Editor for the ensuing year. At that time I made my report upon the Presession Volume, and subsequently was able to supervise the issuing of the Transactions for the year 1928. All bills, etc., for this have been forwarded to the Council and are on file presumably in their records, and the back numbers of the Transactions have been turned over to the Secretary.

Early in this year it became evident that the state of my health would not permit me to supervise the publication of the Presession Volume for 1929. I therefore sent in to the Council my resignation as Editor of the Transactions, and received the request that I continue to hold the position, while the actual work would be done by someone else. The volume which was sent to you before this meeting is the result of this plan, and since I had no part in the work I feel at liberty to join the rest

of the members in congratulating the men who prepared it upon the success which has crowned their efforts.

In presenting this brief quasi-report as Editor of the Transactions, I desire to express my thanks to the members of the Academy for the numerous expressions of commendation which have accompanied my re-elections, and to hope that my successor will meet with as much encouragement from the members and will derive as much pleasure in the performance of the work in the future as I have in the past.

Respectfully,

CLARENCE LOEB.

On motion of Dr. Harry S. Gradle, duly seconded, this report was received and filed.

REPORT OF SENIOR MEMBER OF COUNCIL

DR. E. C. ELLETT: There are several matters that the Council wishes to report to the society.

First, in regard to the Presession Volume. You are familiar with the announcement that appeared in the program in regard to this Volume. When the question was put before the Council they were almost evenly divided as to the advisability of continuing the Presession Volume or discontinuing it. The opinions were so evenly divided that we preferred not to act on it, but refer it to the meeting of the whole Academy.

The Secretary has referred to the matter of the Transactions. We have on hand a good many back numbers which the society would like to sell. Those who are interested may apply to the Secretary. After some experience I think we now have a better idea of exactly the number that are needed for the purposes of the Academy, and probably in future there will not be so many left over. Those in excess of our needs for distribution to the members of the Academy will be for sale to approved libraries and universities and other institutions to which it seems proper to distribute them. This list can be secured by application to the Secretary.

The Council has elected Mr. Herbert Tilley, our Guest of Honor, as an Honorary Member of the Academy.

The resignation of Dr. Clarence Loeb as Editor of the Transactions has been accepted with regret. Dr. Loeb, as you have heard from the letter just read, has been compelled to resign this position on account of the condition of his health.

The Research Committee, through the Council, wishes to report satisfactory progress in the research that has been conducted under the auspices of the Academy during the past year. One of the research men has made his report at this meeting in the shape of a paper, and he has submitted three additional items of research upon which he has been engaged, to the Council. The other member who has been doing research, Dr. Kirby, recipient of the fund in Ophthalmology, has not made a report to the Academy this year, but he made reports the two previous years. This year there was not sufficient new material developed to justify more than a report in the shape of a letter. But the work is going on with entire satisfaction to the Council. The Council wishes to report the distribution of funds for next year's work as follows: For research in Ophthalmology, to Dr. Kirby, $2,000.00; for research in Otolaryngology, to Dr. McNally, $2,000.00; and a grant to the Army Medical Museum, on account of the Laboratory of Otologic and Ophthalmic Pathology, $500.00.

The Finance Committee, as you see in the program, have a rather lengthy report referring to their activities, but in spite of the time they have spent in the past year they desire to report progress at this time and ask to be continued. They are not ready to submit a definite plan for the business arrangements of the Academy.

On motion of Dr. Harry S. Gradle, duly seconded, this report was accepted and placed on file.

THE PRESIDENT: The Committee on Arrangements have no report to make, but the Chair wishes, for the society and himself, to thank Dr. Charlton for his most efficient services in connection with the Committee on Arrangements.

REPORT OF PROGRAM COMMITTEE

DR. W. P. WHERRY: The Program Committee met in Atlantic City in January, and the meeting now coming to a close is the report of that committee. The Program Committee for the coming year will meet some time early in January to consider the program for next year, and any member desiring a place on the program, or having something to present, is urged to submit his subject or his ideas to the proper secretary, Dr. Benedict for ophthalmology, Dr. Myers for otolaryngology, or Dr. Gradle,

Secretary for Instruction. It is earnestly hoped that the members will do this as soon as possible since the program is usually constructed early in the year.

There being no objection, this report was accepted and filed.

Report of Committee on Tuning Forks and Tests of Hearing

To the Members of the American Academy of Ophthalmology and Otolaryngology:

It is impossible for any committee to submit new or startling data each year on a subject which has been so much studied as that of the functional tests of hearing. The most important item which has been brought to your attention during the past few years is the matter of the new magnesium alloy forks. A complete series ranging from C-2 (16 double vibrations) to c-5 (4096 double vibrations) is now in practical use. Considerable testing of patients with these forks has given us definite results from which we may draw some conclusions. Among these are:

1. By air conduction. For testing by air conduction magnesium forks serve just as well as the steel ones, with the exception of the highest tones, namely c-4 (2048 double vibrations) and especially c-5 (4096 double vibrations). However, in those cases where it is desirable simply to learn whether the patient is able to hear a tone of that pitch, these magnesium forks serve very well; but where we wish to find out the actual duration of hearing by air for these high tones, steel forks give somewhat better results. The magnesium fork c-5 (4096 double vibrations) is only heard for a short while, but we hope that Mr. Eisenhour, who originally developed this series of forks, will be able to so modify the highest one that the hearing by air conduction will compare very favorably with the steel fork of the same pitch.

2. For bone conduction. Some of the medium pitched magnesium forks do not give quite as satisfactory results as do the steel forks of the same pitch when placed upon the vertex. However, when they are set upon the mastoid, as in the Rinne test, the magnesium forks of such pitches as c-1 (256 double vibrations, and a-1 (435 double vibrations) are just as useful as the steel forks of the same pitch. With magnesium forks there is a difference between the hearing for air and bone of about thirty seconds

or more. The Weber test can be carried out with one of the lower pitched magnesium forks, such as the C (64 double vibrations).

It is a fact that many of the magnesium alloy forks have considerable overtone, but this is also true of the Bezold-Edelmann series when the weights are removed. It is possible to correct this for those forks which have no weights, by simply applying a rather wide rubber band around the prongs near the distal end. These bands do not damp the vibrations to any appreciable degree, but they do eliminate the overtones.

The outstanding advantages of magnesium alloy forks are that they are rust proof, so that while they oxidize, the metal does not peel; and secondly, that they are very light in weight so that prolonged testing with them does not tire the fingers.

We recommend that these forks be given a thorough test, and we believe that they will prove highly advantageous to the otologist.

There is a new item to present from the Riverbank Laboratory: Mr. Eisenhour has devised a fork with resonator attached, the intensity of which may be varied by changing the size of the aperture of the resonator from zero to the maximum at which the resonator is attuned. This resonator has the advantages of durability, purity of tone, and constant output at a given resonator setting. Practically no sound is transmitted through the supports of the resonator to the table, which is not true in the case of the ordinary wooden resonator.

We believe that experimentation with this resonator may prove of considerable value, but we are not prepared to make any definite statement at this time.

Your committee presents this report and recommends that the committee be continued in order to study further these and other matters affecting functional testing of hearing.

Respectfully submitted,

L. W. Dean,
E. G. Gill,
G. W. Mackenzie,
Robert Sonnenschein, Chairman.

October 24, 1929.

On motion of Dr. A. J. Bedell, duly seconded, this report was accepted and the committee continued.

Report of Otolaryngic Board

Dr. T. E. Carmody: As this Board practically originated in this society I think we must feel very proud of it. We had an examining Board before this actual Board was formed. This society has two members on that Board, and it happens that our worthy President is also President of the Board, and our worthy Secretary is Secretary of the Board, and the amount of work those two gentlemen have been able to perform in spite of their duties for this society, is untold.

The American Board for Otolaryngic Examinations was organized as such in 1924. We have held fifteen examinations to date. We have certified 1490. Out of that number 122 passed the examination in Philadelphia last Monday. The percentage of failures of those coming before the Board is 14.01 per cent.

There being no objection, this report was received and filed.

Report of Ophthalmic Board

Dr. W. H. Wilder: I am not the representative of this society on this Board. Dr. Calhoun is the retiring senior member, and in his absence I will ask you to have the next member, Dr. J. M. Patton, present the report.

Dr. J. M. Patton: I am informed that the Board since its organization has conducted twenty-nine examinations. Since our report a year ago, during 1929, we have held three examinations, the first in connection with the meeting of the American Medical Association in Portland, at which eighteen candidates were present. Due to the number of candidates at the examination this month, and for the convenience of the candidates, one examination was held on the 19th for those who were in the vicinity of New York, and one on the 21st for those in the vicinity of Philadelphia. At New York there were thirty-four candidates; at Philadelphia, thirty. Sixty-three candidates have been certificated during the year, bringing the total certification by the Board up to 752.

The Board wishes at this time to thank those who have so kindly and generously assisted in these examinations. But for this very cordial cooperation the work of the Board would have been greatly hampered.

The Board also wishes to emphasize that they are very willing to arrange for special examinations for localities where a sufficient number of candidates may be wishing for an examination. I do not know exactly what a sufficient number would be, but I should think ten or over. It is the desire of the Board to be of as much service as possible to those who are desiring to secure these certificates.

I think the members of the Board have all felt there has been a very encouraging increase of tone in the preparation of those who have presented themselves for examination, and we feel that this is due not only to the increased interest on the part of the candidates, and an increased desire to meet in a direct way the purposes of the Board, but it is in large measure due to the cooperation of you gentlemen who are preparing condidates for active service in our special field. I refer not only to those who are teaching in postgraduate schools, but to those who have influence over young men, or those who may have young men in their offices and are directly influencing them and helping them to meet these requirements, and who feel a very real responsibility in discharging this duty to our younger men.

The next regular examination will be held in connection with the meeting of the American Medical Association in Detroit.

This report was received and filed.

Report of Committee on Publicity and Legislation

Dr. A. J. Bedell: Your Committee on Publicity and Legislation presents its annual report in three divisions: complimentary, critical, and constructive.

Under the first heading we express our appreciation for the cooperation of Dr. Charlton, the competent chairman of the local committee on arrangements; to the Atlantic City Chamber of Commerce; to the Associated Press, and to the local newspapers. The reports of our sessions have been concise, dignified, and devoid of scare-heads.

We criticize and condemn advertisements in medical journals of special hospitals, particularly the statement of fees charged. This we believe to be detrimental to the best interests of the public and of the profession.

Everyone is conscious of the great work done by the Committee on Lye Legislation and the Committee on the Hard of

Hearing. Our work with the lawmakers of the individual states and with the Federal Government has been simplified by their wonderfully efficient efforts.

Your committee feels that the field of usefulness of this great Academy can be enlarged by the close affiliation with an active ophthalmic journal, and with an equally powerful journal devoted to otolaryngology. To that end we recommend the adoption of a resolution asking the Council of this society to enter into an agreement with such journals. Certainly your committee does not have to explain the great benefits to be derived from such association.

On motion of Dr. Allen Greenwood, duly seconded, this report was adopted.

Report of Committee on Golf

Dr. A. M. Alden: My report as chairman of the Committee on Golf will be brief. The report starts with the golf tournament held last Monday afternoon, at which we had a pleasant time—particularly our distinguished guest. Unfortunately, we did not have as many players as last year, and due to that fact we had an increased deficit, which was arranged for. Next year we ask that those who are going to attend, and know they are going to attend, send in notice to the local chairman. It simplifies his work tremendously. This year Dr. Samuel Skillern, who had charge of the local arrangements, was seriously handicapped by not having any adequate idea of how many men would play. It would have made his work very much easier if you men who came with golf clubs, knowing you were going to play, had sent notice to that effect. Please keep that in mind.

This report was received and filed.

Report of Committee on Ophthalmic and Otolaryngic Pathology

Dr. Harry S. Gradle: The following is the report to the American Ophthalmological Society on the work of the Committee on Pathology:

	Eye Specimens Received and Reported	Eye Specimens Diagnosed When Received	Other Items Illustrations, Models, Etc.	TOTAL
For the 7 yrs. Ending December 31, 1927	800	560	1,124	2484
For the year Ending December 31, 1928	177	46	0	223
January to July 1, 1929	170	0	0	170
TOTAL	1,147	629	1,124	2877

The sets of slides for circulation assembled by the laboratory under the auspices of the American Academy of Ophthalmology and Otolaryngology now consist of twelve sets of 100 slides each. These slides will now be loaned to members of the academy upon application to the Curator of the Army Medical Museum, under the following conditions: A deposit of $25.00 is required to insure against breakage or loss, which deposit will be returned upon the return of the slides. The slides may be kept for two months, and this time may be extended at the discretion of the Curator upon written application. Any breakages will be deducted from the deposit at the rate of $2.50 per slide.

During the year three exceptionally interesting cases have been received and indicate in a small way the value of this collection of the pathology of the eye. They are as follows:

(1) One case of spongioblastoma multiforme of the choroid in a patient, age 36 years, primarily diagnosed a glioma of the retina. There were no evidences of tumor elsewhere.

(2) Capillary hemangioma of the iris in a child four years of age showing recurrent hemorrhage.

(3) Two neurofibromas, one involving the ciliary body and iris, the other on the opposite side of the eye and arising at about the entrance point of the ciliary nerve and vessels, there being no other tumors of this kind.

The last-named case is being reported from this collection in conjunction with the surgeon sending in the specimen. The other two cases are being reported by the surgeons.

It is urged that institutions doing pathological work, and individuals whose pathological work is done apart from the Army Museum, send duplicate slides of interesting cases accompanied

by history and diagnosis to the Army Medical Museum, for the purpose of enriching the figures contained in the second column.

Major George R. Callender is here and will tell us something further of this work.

THE PRESIDENT: The Chair takes great pleasure in introducing Major George R. Callender, of the Medical Corps, U. S. A.

MAJOR GEORGE R. CALLENDER: It has been my fortune to be connected with this collection since its inception in 1922. You have heard the statistical report. I think this loan collection offers opportunity for research in proportion to its size, and we are therefore asking for more specimens. It is my purpose not only to ask for more material, but also to give you some light on some of the interesting things which have come up from time to time.

Before going on with that, I want to say that the appropriation which has been given for the last three years ($500.00) has been used for the making up of this loan set. We now have twelve sets of 100 slides each. The time of the personnel at the Museum did not allow of our handling routine work and at the same time making up these sets, so it had to be done by others on over-time work.

I will say here also that there is an unequal distribution with reference to those sending in these specimens. Relatively few of your large membership have contributed. We have to beg pardon of some who have sent in specimens because of the slowness of the work. We handle a lot of other work in the Museum and reports are sometimes delayed if either the technician or myself happen to be away.

One or two things of marked interest I will show by slides. We would be glad to have comments on the loan collection.

(Slides)

As to collecting figures, with the demands on the personnel it becomes increasingly difficult for the relatively small personnel at the Museum to handle it properly. Furthermore, this Academy has started the first national collection of pathologic specimens in the United States so far as I know. It is entirely independent, it belongs to no institution, no school or organization; it is there for the ophthalmologists of the country. You have gotten hold of something and it must be kept up. It would be my recommendation that a committee be appointed to consider ways and

means of being sure that this collection continues and builds up to the maximum value.

Dr. Verhoeff and Dr. Friedenwald examine these cases and the reports are either formulated by them or verified by them. You are getting excellent service in the way of pathology.

There being no objection, this report was received and placed on file.

REPORT OF THE COMMITTEE ON OPTICS AND VISUAL PHYSIOLOGY

DR. W. H. WILDER: This Committee on Optics and Visual Physiology has had several meetings and has deliberated considerably over various topics. It has not heretofore made any very definite or concrete suggestions as to the course of procedure, and we are still considering some rather vital subjects. We wish, however, on this occasion to present for your consideration, and also we hope for your endorsement, the following proposition relative to something like symmetrical notation of vision. We know that some ophthalmologists are using the metric system, some the old foot system, so far as recording the distance vision is concerned, and some are even using the notation 1/10, 2/10, 3/10. We think now is the time for some official body to take action looking toward a uniform plan of notation. We therefore propose to launch out now and secure some uniformity in the matter and resolve that we will all use the same method. It will be a little difficult at first, but when we come to some understanding about it I think we will have a more successful notation.

Your Committee therefore recommends for your consideration and endorsement the following:

We recommend that all visual acuity notations for distance and near be made in the metric system. To carry out this recommendation a sub-committee had been appointed to enlist the cooperation of the Bureau of Standards in Washington in the preparation of test charts for distance and near with notations in the metric system.

Your Committee further recommends to the program committee that at least one paper in the scientific program at every meeting be devoted to some subject closely related to visual physiology or optics.

There being no objection, this report was received and placed on file.

Report of Committee on Lye Legislation

Dr. Joseph C. Beck: The Committee has no report to make, but in view of the importance of the subject, and in appreciation of Dr. Jackson's work and effort in this matter, we would ask you to continue this committee so that by next year we may perhaps have some additions to make to the report.

On motion of Dr. Horace Newhart, duly seconded, this report was adopted and the committee continued.

Report of the Committee on the Hard of Hearing

Your Committee deeply regrets the loss of one of its most valued members in the recent death of Dr. Charles W. Richardson. He was a most active worker, a wise counsellor, and a real friend of the deaf and hard of hearing in our country. With the approval of your President, Dr. Austin A. Hayden of Chicago has been chosen to fill this vacancy.

During the past year your Committee has carried on various activities consistent with its purpose. It interprets as one of its important functions the dissemination among the Fellows of the Academy of information concerning the progress of efforts made in behalf of the hard of hearing; also the giving to the laity fundamental medical facts relating to the prevention of deafness and the conservation of hearing. Acting in this capacity, papers on the use of the audiometer among school children have been presented by members of this committee before the Section on Laryngology, Otology and Rhinology of the American Medical Association, the American Association of School Physicians, the Child Hygiene Section of the American Public Health Association, and various other state and local organizations.

Under the joint auspices of the American Medical Association and the Chicago Laryngological and Otological Society, an address on the Prevention of Deafness was broadcast during National Hearing Week. Several members of your committee participated in the Second Conference on the Deaf and Hard of Hearing, called by the Division of Psychology and Anthropology of the National Research Council in Washington in February, 1929. The ultimate result of this conference promises large benefits to this handicapped group.

Your Committee is actively interested in promoting the formation of a national coordinating committee on the Prevention of

Deafness and the Conservation of Hearing, this committee to be organized upon the invitation of the American Medical Association but bringing into closer contact not only our societies composed of otolaryngologists, but securing the cooperation of other interested national groups.

Due largely to the influence of one of our members, the Chicago Laryngological and Otological Society recently evidenced its interest in the cause of the deafened by voting to take out membership in the Chicago League for the Hard of Hearing for all of its members—an example which might well be followed by other local groups of otolaryngologists.

During the past year there has been a notable growth in the periodic testing of hearing acuity of public school children by means of the audiometer. The work has already been started in more than one hundred cities. A survey of these activities recently undertaken by your committee shows that the work is already well established in thirty-four cities with an aggregate population of 6,800,000—a remarkable record in view of the fact that the movement is only three years old. Our survey reveals the urgent need of careful leadership and oversight in this special work such as can be given only by the trained otolaryngologist. It is a field of public health work and should have the fullest cooperation of the medical man and should not be left to others.

Public interest in the accurate periodic testing of the hearing of persons of all ages is growing rapidly. Such examinations will soon constitute an important part of every comprehensive health program, be it for the individual or for the large group, particularly our public school children. To meet promptly this growing demand your committee, after due deliberation, respectfully recommends that every member of this society offering to the public his services in the special field of otology adequately equip himself for the most accurate diagnostic work in the detection of hearing impairment.

Your Committee is carrying on a survey to determine the relative incidence of defective hearing among the neglected school children of rural communities, and the children of larger cities, where applied school hygiene has for years been in effective operation.

Your Committee recommends its continuance for the further study of problems it has undertaken.

Respectfully submitted,

HOWARD V. DUTROW,
HAROLD HAYS,
AUSTIN A. HAYDEN,
GEORGE L. TOBEY,
CLAUDE T. UREN,
HORACE NEWHART, Chairman.

October 24, 1929.

On motion of Dr. J. M. Patton, duly seconded, this report was accepted and the committee continued.

REPORT OF COMMITTEE ON YEAR BOOK

DR. HARRY S. GRADLE: Your Committee on Year Book was appointed several years ago in the hope that ways and means might be found for perpetuating the Ophthalmic Year Book, but in that respect the committee has been unsuccessful. The committee wishes to report absolute failure and asks to be discharged.

On motion of Dr. Gradle this report was accepted and the committee discharged.

REPORT OF COMMITTEE TO INVESTIGATE THE TREATMENT OF INCIPIENT CATARACT

The Chairman of this committee, Dr. Allen Greenwood, presented the following letter from Dr. Kirby as the report of this committee.

October 3, 1929.

During the past year a further study of the natural history of untreated cataract and the means by which one may judge of its progress or arrest has been made. Two years ago a report was made before the Academy in which the matter of standards for this judgment was given thorough consideration. At the present time it is felt that these standards still hold good and that data secured in this way by periodic examinations of cataract patients will be of considerable value in the future. These tests related to standards connected with: 1. Vision and changes in vision. 2. Refraction and changes in refraction. 3. Ophthalmoscopy. 4. Bio-microscopy.

The matter of having the patient draw his subjective impression of the lens opacity and record its changes at stated intervals has also been considered. It was judged, however, that

this would have too great an effect in making the patient introspective over his condition. Dr. J. H. Evans suggested that it might be possible to record the changes in the lens by measuring the time relationship between light-exposure of the retina and the after image. Changes in the refractive media influence this time relationship. Sufficient work in this has not been done.

In regard to photography, a binocular camera was secured to be used in connection with a slit-lamp microscope, in order to work out a satisfactory technique. The great advances which have been made in photography would make it seem likely that the camera will serve a most useful purpose in recording changes in lens opacities. It will be necessary to be able to show the very fine details of incipient cortical and nuclear cataract, such as a lamellar separation, water-splitting of the sutures, vacuoles, and nuclear haze. At the present time there is still great difficulty encountered with the normal relucency and efflorescence of the lens when direct light falls upon it. This obscures for photographic purposes the very fine details of incipient cataract. Besides, it is a well established fact that the transparency of any medium should be judged by transmitted light. There is at present no technique available for the use of transmitted light for photography of the crystalline lens. However, it is hoped that these obstacles may be overcome so that the camera will serve a more useful purpose in this research.

A well-known firm has supplied a quantity of lens extract and parathyroid extract for use in our research. The experiments with this material are still being carried on.

DANIEL B. KIRBY.

REPORT OF SECTION ON OPHTHALMOLOGY

DR. W. L. BENEDICT: The Secretary wishes to report that the program as printed is the result of our efforts for this year and will stand for what it is worth. We wish to say that this program was built with the Presession Volume in mind. The authors were told to send their papers in in time so the members of the Academy could read them in the Presession Volume, and that then they should present their papers before the Academy in abstract. I bring this to your attention now because of the question you will be called upon to consider in connection with the Presession Volume. In my opinion, if the Presession Volume is discontinued an entirely different type of program should be prepared for next year.

There being no objection, this report was accepted and filed.

Report of Section on Otolaryngology

Dr. John Myers: This year, as far as possible, the committee has arranged a program with continuity of thought. In doing this some members have been disappointed at not having a place on the program. However, we felt it was better to have some subjects fully developed, as has been done in the symposium on Brain Abscess, and on the Ethmoid, than to have various subjects that had no connection with each other. By carrying out the plan of having at least two subjects thoroughly discussed each year, our Transactions are rendered the more valuable for reference.

Much energy was expended in working out the program before you. Every subject submitted by members had the careful consideration of each man on the committee, and with great reluctance many subjects that ought to be presented to the Academy were deferred to another time. While there was much work in building this program, it was made most pleasant because of the cooperation of all with whom we had correspondence. As a rule our letters were answered promptly and a spirit of good will was always manifest.

This report would not be complete, however, without expressing our opinion as to the advisability of continuing the publication of the Presession Volume. For two years we have tried the experiment, and in our opinion it has not proven a success.

1. It is most difficult for essayists to prepare their papers for publication two or three months before they are to be read, as they want the latest data therein.

2. While it is a pleasure to keep reminding the authors that the time is near, has approached, or is past, for papers to be in the hands of the publishers, it must be most exasperating to busy men to be thus constantly reminded; and yet even when this is done it has not always been possible to get all papers in the Presession Volume.

3. Often the papers published in the Presession Volume have to be revised very much for the Transactions, and this is an added expense.

4. The Presession Volume costs too much to justify continuing the experiment.

In closing, we wish to thank everyone who has assisted us during the last year by making helpful suggestions and con-

structive criticism, and we trust that next year you will feel free to give to the secretarial force that loyal support and wholehearted cooperation that you have given during the past year.

There being no objection, this report was accepted and filed.

Report of Section on Instruction

Dr. Harry S. Gradle: The report of the Secretary on Instruction is the instructional courses that we are having Wednesday, Thursday and Friday of this week. The Section on Instruction is for the Academy, and it is our desire to follow the wishes of the members in these courses. I must confess that we have received very little help in making out our program despite repeated inquiries regarding suggested courses. Still we will try to struggle along the best we can and try to give you as nearly what you want as we can estimate.

This year, at the suggestion of Dr. Mosher, we have added two afternoon courses dealing with the progress in our specialties during the last few years—one afternoon devoted to otolaryngology and one to ophthalmology. The first talk this afternoon seemed to be well received, sufficiently that it is deemed wise to continue this for another year or so at any rate, until the members decide definitely whether or not they wish these lectures.

Next year we wish to try another plan, following Dr. Kopetsky's suggestion—have an afternoon session at which teachers, both of graduate and undergraduate schools, may assemble most informally for discussion of the problems of teaching. We believe if we put that into the program there may be a sufficient number interested to justify this informal, purely voluntary assemblage.

In concluding this report let me ask for written suggestions as to the courses in instruction—the length of time of the courses, whether we should have so many subjects, so that we may not increase the courses beyond all rhyme or reason.

There being no objections, this report was accepted and filed.

Report of Committee on Necrology

The following members of the American Academy of Ophthalmology and Otolaryngology have died since our last meeting:

Dr. James Moores Ball, St. Louis, Missouri.
Col. J. M. Banister, Omaha, Nebraska.
Dr. Robert Vance Brawley, Salisbury, North Carolina.
Dr. Eldred Beverly Cayce, Nashville, Tennessee.
Dr. Breese Morse Dickinson, Pittsburgh, Pennsylvania.
Dr. Joshua A. Ellegood, Wilmington, Delaware.
Dr. Arthur E. Ewing, St. Louis, Missouri.
Dr. C. G. Fellows, Chicago, Illinois.
Dr. Clarence Leslie Frey, Scranton, Pennsylvania.
Dr. Edward Ford Gavin, Waukegan, Illinois.
Dr. Otto Glogau, New York, New York.
Dr. F. B. Harding, Allentown, Pennsylvania.
Dr. Thomas Corwin Hood, Indianapolis, Indiana.
Dr. Lucien Howe, Buffalo, New York.
Dr. Leon Webster Jessaman, Framingham, Massachusetts.
Dr. Darwin Mills Keith, Rockford, Illinois.
Dr. J. S. Kirkendall, Ithaca, New York.
Dr. Lafayette Page, Indianapolis, Indiana.
Dr. C. S. Rebuck, Harrisburg, Pennsylvania.
Dr. Charles W. Richardson, Washington, D. C.
Dr. Martin P. Rindlaub, Fargo, North Dakota.
Dr. E. L. Russell, Kansas City, Missouri.
Dr. William Hardin Sears, Huntingdon, Pennsylvania.
Dr. Greenfield Sluder, St. Louis, Missouri.
Dr. Myles Standish, Boston, Massachusetts.
Dr. Lewis H. Taylor, Wilkes-Barre, Pennsylvania.

Dr. James Moores Ball

Dr. James Moores Ball, nationally known oculist and writer on medical subjects, died March 1, 1929, at the age of sixty-six years, of heart disease. He had been ill for ten days.

Best known for his book, "Ball's Modern Ophthalmology," Dr. Ball was prominent in American and European medical circles and was an honorary member of many European professional societies.

He is survived by his widow, Mrs. Namoi Marshall Ball; a son, Logan Marshall Ball; a daughter, Mrs. Robert Lafean of York, Pennsylvania, and three brothers, Dr. Otho F. Ball of Chicago, and Frank and Malvin Ball of Saskatchewan, Canada. Dr. Otho Ball is publisher of the Modern Hospital Magazine and the Nation's Schools.

By his own wish, Dr. Ball's body was cremated and the ashes placed in the Army Medical Museum in Washington, to which, in 1922, he donated his collection of anatomical specimens, drawings, charts, and casts. The collection, made over a period of thirty years, is said to be second only to that of Dr. George E. de Schweinitz of Philadelphia.

Besides his work on ophthalmology, Dr. Ball wrote two other volumes, "Andreas Vesalius, Reformer of Anatomy," issued in 1910, and "The Sack-'Em-Up Men," an account of the days when graveyards were looted for human dissection specimens. The latter book was issued last year.

Recently, Dr. Ball presented a valuable collection of old and rare medical books to the St. Louis Medical Society, about 350 volumes in all. Some of the volumes are several hundred years old. They are printed in many languages.

Dr. Ball was born in West Union, Iowa. He received his medical degree from the University of Iowa in 1884, and took several postgraduate courses in New York and Europe.

Later he became Professor of Diseases of the Eye at National University here, and was oculist in National University Hospital. For a time he was editor of the Annals of Ophthalmology and served as Professor of Eye Diseases at the St. Louis College of Physicians and Surgeons.

In 1890 Dr. Ball married Miss Naomi Marshall in York, Pennsylvania.

Originally engaged in the general practice of medicine, Dr. Ball, in 1890, confined his practice to ophthalmology. He practiced in St. Louis for about thirty-five years.

Colonel J. M. Banister

Colonel J. M. Banister, Medical Department, U. S. Army (Retired), was born at Greensboro, Alabama, and died January 31, 1929, at the age of seventy-four years.

He was the son of an Episcopal minister. In 1874 he graduated from Washington and Lee University and then attended the University of Virginia Medical College and received his degree in 1878. He entered the Medical Corps of the United States Army in 1879 and served with distinction until his retirement, after over thirty years' service, in 1910.

During his service he developed his knowledge of opthalmology by studying in New York and Philadelphia. While stationed at Fort Leavenworth he carried out a series of tests on healthy soldiers which established a new standard in regard to the dynamics of coordinated eye movements. He early recognized the value of the Bassini operation, and it was through his efforts that it was introduced into the Army and became the authorized procedure for hernia. When in command of the hospital at West Point he modernized and equipped the operating room, and used his operative skill to give the cadets the benefit of skilled, modern surgery. In the Philippines, while in command of the Department of Luzon, and later of the First Reserve Hospital, he met the problems of tropical medicine fearlessly and successfully. Wherever he served, in whatever situation, he fulfilled his duties with intelligence, courtesy, skill, and courage.

Dr. Banister was President of the Nebraska State Medical Society in 1919.

Dr. Robert Vance Brawley

Dr. Robert Vance Brawley was born November 9, 1878, at Shepherds, North Carolina, the son of J. W. and Elizabeth Brawley. He died January 5, 1929.

He graduated from the University of North Carolina, Chapel Hill, North Carolina. He also was a graduate of Medical College, Richmond, Virginia, and later took postgraduate work at the New York Eye, Ear, Nose and Throat Hospital. He came

to Salisbury, North Carolina, and began to practice in 1903, doing eye, ear, nose and throat work. He was appointed oculist for the Southern Railway Company in October, 1905.

Dr. Brawley was married to May Boyden, October 23, 1907, who, with three sons, survive him.

Dr. Eldred Beverly Cayce

Dr. Eldred Beverly Cayce died suddenly January 4, 1929.

He received his education in Battle Ground Academy, Franklin, Tennessee, and his degree of M. D. from the University of Nashville in 1910.

He did postgraduate work in Vienna and London in 1911.

He was a member of the Tennessee State Medical Society, Southern Medical Association, American College of Surgeons, and the American Academy of Ophthalmology and Otolaryngology.

Breese Morse Dickinson, M. D., F. A. C. S.

Doctor Breese Morse Dickinson, born April 4, 1871, in Brown County, Kansas, died unexpectedly at his home in Pittsburgh, Pennsylvania, on April 11, 1929, from a vascular lesion of the brain, presumably a thrombosis.

After completing his academic curriculum at the University of Kansas, he received his doctorate in Medicine from the University of Pennsylvania in 1899. Journeying to Pittsburgh, Pennsylvania, the same year, he interned at the Mercy Hospital. For more than twenty-five years he was a member of the staff of this institution, and for over fifteen years was the Senior Otolaryngologist.

He was a member of the American Academy of Ophthalmology and Otolaryngology, American College of Surgeons, Pittsburgh Academy of Medcine, American Medical Association, and the Allegheny County Medical Society.

Dr. Joshua A. Ellegood

Dr. Joshua A. Ellegood, of Wilmington, Delaware, died April 2, 1929, in Florida, of myocarditis, at the age of seventy years.

Dr. Ellegood received his M. D. degree from Jefferson Medical College of Philadelphia in 1881.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and the American Laryngological, Rhinological and Otological Society.

He was on the staff of the Delaware Hospital.

Dr. Arthur Eugene Ewing

Dr. Arthur Eugene Ewing was born in Cartersville, Georgia, April 6, 1855. He was graduated from Dartmouth College in 1878; studied law and was admitted to the Alabama Bar in 1879. Finding the legal profession not to his liking, he turned to medicine, his father's profession, and was graduated from the Saint Louis Medical College in 1883. His medical education was completed by three years' training in the office of Dr. John Green, followed by two years at the Koenigliche Christian-Albrechts University at Kiel, Germany. He then returned to St. Louis and became associated with Doctors Green and Post.

From that time until his death he was connected with Washington University, being appointed Professor of Ophthalmology in 1902 and retaining this chair until 1921, at which time he became Professor Emeritus.

Dr. Ewing received the degree Master of Arts from Washington University in 1912, and Doctor of Science in 1926. He was at one time Vice-President of the Section on Ophthalmology of the American Medical Association.

Dr. Ewing combined, to an unusual degree, the clinician and the scientist, taking care of a very large practice and yet producing much original work. Among his contributions were a very successful lid operation for trichiasis and entropion; test letters and optotypes; trachoma forceps; lid specula; double fixation forceps; "bread-knife" capsule forceps; and lid clamps.

Still extremely busy with his practice and operating with a steady hand in his seventy-fourth year, he suffered a cerebral hermorrhage on January 23, 1929, from which, three days later, he died.

Among the last of the older group of ophthalmologists in St. Louis, Dr. Ewing was unquestionably for years the dean of the profession in that community. His counsel was sought by his ophthalmological confreres, as well as his patients, for his excellent judgment, based on wide experience, was well known. His kindly nature made him loved by all and placed him among those whose passing is most regretted.

Dr. C. Gurnee Fellows

Dr. C. Gurnee Fellows was born in Milwaukee, Wisconsin, April 27, 1863, and died February 2, 1929.

He received his A. B. degree at Lawrence College, Appleton, Wisconsin, in 1883; his A. M. degree in 1886; and his M. D. degree at Hahnemann Medical College, Chicago, Illinois, in 1885. Later he took postgraduate work in New York, Vienna, Paris, and London. He practiced in New Orleans from 1885 to 1888, and in Chicago from 1889 until his death.

Dr. Fellows was formerly Senior Professor of Ophthalmology and Otology at Hahnemann Medical College, and Senior Attending Surgeon of the Eye and Ear Department of the same college.

He was a member of the American Institute of Homeopathy; Illinois Homeopathic Medical Association; Chicago Homeopathic Society; American Homeopathic Ophthalmological, Otological and Laryngological Society, of which he was President in 1892 and 1893; American Medical Association; Illinois State, and Chicago Medical Associations; Fellow of the American College of Surgeons; member of the American Academy of Ophthalmology and Otolaryngology.

His widow, a son and a daughter survive him.

Dr. Clarence Leslie Frey

Dr. Clarence Leslie Frey, the son of a physician, was born at Glen Rock, York County, Pennsylvania, November 7, 1851, and died on March 13, 1929, at the age of seventy-eight.

In 1870 he entered Jefferson Medical College at Philadelphia, and graduated in March, 1872. Soon after this he became one of the physicians at the Danville Hospital for the Insane, where he remained for several years. Later, he pursued a course of study for two years in Vienna, Austria, after which he returned to Scranton and opened offices as an oculist.

Dr. Frey was one of the oldest and most faithful members of his local society. He always displayed a keen interest in its affairs, and at one time served as its President.

By his professional ability and his devotion to duty he had built up a large practice, and was highly esteemed and beloved by his patients.

Dr. Edward Ford Gavin

Dr. Edward Ford Gavin was born in Waukegan, Illinois, in 1864, and died March 23, 1929.

He was educated in the public schools of Waukegan, and graduated from Rush Medical College in 1890. Was an interne at St. Luke's Hospital.

He first began the practice of medicine in Waukegan in 1891 and afterwards became local surgeon to the Chicago and Northwestern Railroad Company.

Dr. Otto Glogau

Dr. Otto Glogau, of New York City, died March 6, 1929.

He received his M. D. degree from the University of Vienna in 1900. He was a Fellow of the New York Academy of Medicine; a Fellow of the American Medical Association; a Fellow of the American Academy of Ophthalmology and Otolaryngology; and a members of the Otolaryngology Committee.

Dr. Frederick B. Harding

Dr. Frederick B. Harding died January 8, 1929. He was born in Shickshinny, Pennsylvania, attended the public schools of Milton, and was graduated from the high school with honors in 1902. He attend Dickinson College for one year and then transferred to Bucknell University, where he received his degree three years later. He graduated from Medico-Chi. College in Philadelphia, class of 1908. He was an interne in the State Hospital at Scranton, and then went to Tamagua, where he conducted a practice until the entrance of the United States into the World War. He served in the United States Forces as a Captain in the Medical Corps.

At the close of the World War, Dr. Harding went to Marseilles, France, for special medical study. After studying in Marseilles, Doctor Harding returned to the United States and located in Allentown, limiting his practice to the eye, ear, nose and throat.

Besides being a member of the Allentown Order of Elks, he was a 32nd degree Mason and a member of the American Legion.

Dr. Harding was married on October 6, 1928, to Mary Troxell. Besides his parents, his widow is the only survivor.

Dr. Thomas C. Hood

Dr. Thomas C. Hood was born in western Indiana, October 28, 1860. He was educated in the grade and high schools, entered Wabash College in 1881, and graduated from that institution four years later.

Dr. Hood entered Jefferson Medical College in 1885, and after graduation practiced general medicine for a number of years. Deciding to specialize in diseases of the eye, he then did extensive graduate work in New York, Berlin, and Vienna. Following these special studies he located in Indianapolis, where he practiced ophthalmology exclusively until the date of his death from influenza, January 2, 1929.

Dr. Hood was well known, both locally and nationally, as an oculist. He was Professor of Ophthalmology in the Indiana University School of Medicine for several years, but had recently retired from teaching. He was a member of the Indianapolis Medical Society, the Indiana State Medical Association, the Indiana Academy of Ophthalmology and Otolaryngology, the American Medical Association, the American Academy of Ophthalmology and Otolaryngology, and the American College of Surgeons.

Mrs. Hood and one son survive.

Dr. Lucien Howe

Dr. Lucien Howe, ophthalmic surgeon, was born in Standish, Maine, September 18, 1848, and died at his home in Belmont, Massachusetts, December 27, 1928, aged 80. He was the son of Marshall S. and Anna (Cleland) Howe.

Dr. Howe was educated at Bowdoin, A. B., 1870; A. M., 1873; M. D., Long Island Medical Hospital, Brooklyn, 1871; M. D., Bellevue Hospital Medical College (New York University), 1872; M. C. R. S., London, 1873.

He was married, August 16, 1893, to Elizabeth M. Howe of Cambridge, Massachusetts, and resided in Buffalo, New York.

He was Surgeon-in-Charge of the Buffalo Eye and Ear Infirmary since 1876; Professor of Ophthalmology, University of Buffalo, 1879 to 1919, then Professor Emeritus; Ophthalmological Surgeon, Buffalo General Hospital, since 1885.

Dr. Howe was a Fellow of the Royal Society of Medicine; Fellow, Medical Society of the State of New York (Vice-Presi-

dent); member Ophthalmologische Gesellschraft; of Societe Francaise d'Ophthalmologie; of Ophthalmological Society, United Kingdom; of American Ophthalmological Society (ex-President); American Medical Association (Chairman Committee on Prevention of Heredity Blindness, Section on Ophthalmology; Chairman Committee on Investigation Concerning the Ocular Muscles, Section on Ophthalmology).

In May, 1926, Dr. Howe established a Foundation known as "The Howe Laboratory of Ophthalmology" at Harvard University, for ophthalmological research, and turned over to the Trustees of Harvard University the sum of $250,000.00 for this endowment. Subsequently, the Board of Incorporators of Harvard University officially selected Dr. Howe as the Director of Research under this Foundation.

In 1927 he was presented with the Leslie Dana Medal, in appreciation of his services in Law, Science and Medicine for the prevention of blindness.

Dr. Leon Webster Jessaman

Dr. Leon Webster Jessaman of Framingham, Massachusetts, died June 11, 1929, at the age of fifty years.

He graduated from Tufts College Medical School at Boston, Massachusetts, in 1909, and was on the staff of the Framington Union Hospital, Framingham, Massachusetts, up to the time of his death.

Dr. Jessaman was a member of the American Academy of Ophthalmology and Otolaryngology, the New England Ophthalmological Society, and New England Otological and Laryngological Society.

He served in the Medical Department during the World War.

Dr. Darwin M. Keith

Dr. Darwin M. Keith, aged 62, died at his home in Rockford, Illinois, June 8, 1929, after a brief illness of pneumonia. He was born in North Fairfield, Ohio, October 15, 1866, the son of Dr. Darwin Mitchell Keith and Caroline Mills Keith.

His early education was received in Ohio and New York. He entered Oberlin College when so young that sponsorship of a professor was necessary for his entrance. He later attended the University of Michigan and Columbia University, and after-

ward studied abroad at St. Bartholomew's College, London, and at the University of Brussels. He received his degree in Medicine at the Belgian institution.

Postgraduate study, preparing him for specialization in eye, ear, nose and throat work, was taken by Dr. Keith in London, Vienna, Paris, Berlin, and Copenhagen. He had made a practice for many years of returning to Europe at frequent intervals for further study.

Dr. Keith headed the Rockford Hospital eye, ear, nose and throat staff, and was a member of the Winnebago County Medical Society. He attained many honors, both at home and abroad, in his profession, and was a member of the Royal College of Surgeons, a Fellow of the American College of Surgeons, a member of the American Academy of Ophthalmology and Otolaryngology, of the Royal Aberiathin Society of England, and of the American Medical Association. At one time Dr. Keith was Assistant Laryngologist at the St. Bartholomew and Golde Hospitals, London.

During the World War, Dr. Keith joined the colors and was commissioned a captain in the Medical Corps. Upon the cessation of hostilities he accepted a Major's commission in the Medical Reserve Corps, which he held at the time of his death.

Dr. J. S. Kirkendall

Dr. John S. Kirkendall, of Ithaca, N. Y., died at his residence at the age of seventy-two. Death resulted from a skull fracture sustained in a fall downstairs at his home, a few days previous.

He was a member of the American College of Surgeons, Fellow of the New York Academy of Medicine since 1893; Fellow of the American Laryngological, Rhinological and Otological Society since 1906; member of the American College of Ophthalmology, Fellow of the American Academy of Ophthalmology and Otolaryngology and member of the Buffalo Ophthalmological Society since 1897.

Dr. Kirkendall was attending opthalmologist of the Willard State Hospital for over 30 years, the first specialist in this field to receive state appointment. He was consulting ophthalmologist for the Ithaca Memorial Hospital, the Reconstruction Home, and the Lehigh Valley Railroad.

Dr. Lafayette Page

Dr. Lafayette Page was a native of Kentucky, where he lived until early manhood and where he received his preliminary education. He came to Indianapolis in 1884, entered Indiana Medical College in 1885, and graduated in Medicine from that institution in 1888. Following this he spent one year in the general practice of medicine in the Indianapolis Dispensary. Then he determined to take up the rather new specialty of otolaryngology, and to prepare himself adequately for this practice spent one year in the study of ear, nose and throat in the leading clinics of Europe. Upon his return to America he located in Indianapolis, where he at once secured a lucrative practice which he maintained until his death in July, 1929, at the age of sixty-six.

Dr. Page was a member of the Indiana State Medical Association, the Indiana Academy of Ophthalmology and Otolaryngology, the American Academy of Ophthalmology and Otolaryngology, the American Rhinological, Laryngological and Otological Society, the American Bronochoscopic Society, the American College of Surgeons, the Indianapolis Medical Society, and the American Medical Association. He was for many years Professor of Otolaryngology in Indiana University School of Medicine, with which institution he maintained close relationship until his death.

Dr. Page served as President of the Indianapolis Medical Society and as Vice-President of the American Rhinological, Laryngological and Otological Society. He also served in Base Hospital No. 32 during the World War, receiving the rank of Major. In 1927 Indiana University conferred upon him the degree Doctor of Laws.

He is survived by his widow, two sons, Lafayette Page, Jr., and Dr. Irvine Page, and one daughter, Mrs. Thomas Fisher of Chicago.

Dr. Charles Stough Rebuck

Dr. Charles Stough Rebuck was born October 18, 1873, in Harrisburg, Pennsylvania, and died September 9, 1928. He received his early education in private schools and in the public schools of Harrisburg, was a graduate of the Cumberland Valley State Normal School, and studied medicine at the College of Physicians and Surgeons, Baltimore, a graduate of the class of 1896. The same year he began the practice of medicine in Har-

risburg, Pennsylvania. Later, he became an eye, ear, nose, and throat specialist, preparing for this work by postgraduate courses in the leading hospitals in this country and in London, England.

He was a member of the staff of the Harrisburg Hospital for twenty-three years, consulting specialist of the State Hospital for the Insane at Harrisburg, laryngologist for the State Sanatoria at Mount Alto and Hamburg for a number of years, and consulting surgeon of the Annie Warner Hospital, Gettysburg, Pennsylvania, and also eye specialist to the Pennsylvania Railroad.

Dr. Rebuck established medical inspection in the public schools of Harrisburg, and the system now in use may be credited to his work in what was then a new field, only a few states at that time having medical inspection in schools.

Dr. Rebuck was a Fellow of the American College of Surgeons, a member of the American Academy of Ophthalmology and Otolaryngology, of the American Medical Association, the Medical Society of the State of Pennsylvania, the Dauphin County Medical Society, and the Harrisburg Academy of Medicine.

Dr. Rebuck enlisted in the World War and served with the rank of Captain in reconstruction hospitals.

Dr. Charles W. Richardson

Dr. Charles W. Richardson, sixty-nine years of age, died August 25, 1929, in a Boston hospital, from complications following an operation three weeks before, for an intestinal trouble.

Dr. Richardson was ear, nose and throat specialist on the medical staff at the White House during both the Wilson and Coolidge administrations.

One of his most recent contributions to science was the completion of an extended national survey of institutions for the deaf, under the auspices of the National Council to determine the physical causes of deafness. He was chairman of a special committee, composed of leading specialists, named to direct the survey.

Dr. Richardson was born in Washington on August 22, 1861, the son of Charles F. E. and Charlotte Ann Williamson Richardson. He was a student at Columbian, now George Washington, University from 1878 to 1880. He received the degree Doctor

of Medicine from both George Washington and the University of Pennsylvania in 1884.

He was Professor of Laryngology and Otology at George Washington from 1891 to 1921, and was given the honorary degree Doctor of Science by that institution at the conclusion of his long period of service. This same degree was also conferred upon him in 1927 by the University of Pennsylvania.

During his career Dr. Richardson was president of various medical organizations, among which were the American Laryngological, Rhinological and Otological Society, the American Otological Society, and the District of Columbia Medical Society. At his death he was serving as a Trustee of the American Medical Association, to which position he was first elected in 1921.

During the World War he served in the Medical Corps, in which he rose to the rank of Colonel. He was first commissioned a Major in the Medical Reserve Corps.

Dr. Richardson made many valuable contributions to textbooks on diseases of the ear, nose and throat. The books included Posey and Wright's Diseases of the Eye, Ear, Throat and Nose; Reference Handbook on Medical Sciences; and Musser and Kelley's Handbook of Practice and Treatment.

Dr. Martin Philip Rindlaub, Jr.

Dr. Martin Philip Rindlaub, Jr., was born November 6, 1874, at the city of Platteville, Wisconsin, and died December 2, 1928, at Minneapolis, Minnesota.

He graduated from the University of Wisconsin in 1896 with the degree of B. L. He entered Johns Hopkins University as a student of medicine and graduated therefrom with honors in 1905 with the M. D. degree.

He held membership in the Cass County, North Dakota, Medical Society, and in 1911 he was Secretary and in 1912 President of that organization. In 1925 he was President of the North Dakota Academy of Ophthalmology and Otolaryngology. He was also a member of the Minnesota Academy of Ophthalmology and Otolaryngology, a member of the North Dakota State Medical Association, a member of the American Academy of Ophthalmology and Otolaryngology, and a Fellow of the American College of Surgeons. He was also nationally known in college fraternity circles, and was prominent in the affairs of his fraternity, Sigma Chi.

He enlisted in the service of his country on July 10, 1918, and was honorably discharged on June 13, 1919. He served as Captain in the Medical Corps of the United States Army and was a member of the Medical Board of Review at Camp Grant, Illinois.

Dr. Edwin L. Russell

Dr. Edwin L. Russell of Kansas City, Missouri, died December 19, 1928, at the age of fifty-five years.

He was a graduate of the public schools of Kansas City, and of the University Medical College, Kansas City, class of 1901. He took a postgraduate course in New York in 1920 and studied abroad at Budapest and Vienna in 1924.

Dr. Russell was a member of the Jackson County Medical Society; Fellow of the American Medical Association; Fellow of the American Academy of Ophthalmology and Otolaryngology; and charter member and Secretary of the Kansas City Society of Ophthalmology and Otolaryngology.

He left surviving, his father, four sisters and one brother.

Dr. William Hardin Sears

Dr. William Hardin Sears was the son of Rev. Samuel W. and Elizabeth P. Sears, and was born in Cumberland, Maryland. He died June 14, 1929, aged fifty-nine.

He entered Medico-Chirurgical Medical College in Philadelphia, from which he graduated with the degree of Doctor of Medicine in 1898. For two years thereafter he was resident surgeon of Philadelphia General Hospital, and in 1900 did postgraduate work at Polyclinic and other hospitals of Philadelphia. He also studied in Vienna, Sweden, Bordeaux, France, and at the University of Pennsylvania.

Dr. Sears was a member of the Huntingdon County Medical Society, the Medical Society of the State of Pennsylvania, American Medical Association, Fellow of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American Laryngological, Rhinological and Otological Society, member of the American Otological Society, former chairman of the Section on Eye, Ear, Nose and Throat, Medical Society of the State of Pennsylvania. He held a certificate of the American Board of Ophthalmic Examinations, and the same from the American

Board of Otolaryngology. He was a Fellow of the American College of Surgeons, member of the Oxford Ophthalmological Congress, Oxford, England.

For the past few months, Dr. Sears had been giving a series of lectures, by invitation, at the University of Pennsylvania, on special affections of the eye and ear.

He was a member of the staff of the J. C. Blair Memorial Hospital, Surgeon-in-charge of the eye, ear, nose and throat department of that institution; also at the Pennsylvania Industrial Reformatory and Home for Friendless and Orphaned Children. He was consulting ophthalmologist at the Lewiston Hospital. He had served as a trustee of the J. C. Blair Memorial Hospital since its organization.

He was the author of many treatises, books and case reports. Among his published works may be named the following:

"The Ophthalmologist in Diagnosis of Epidemic Encephalitis"

"The Gradenigo Syndrome with Report of Three Cases"

"Practical Use of the Barany Tests Away from Medical Centers"

"Otogenic Paralysis of the Abducens"

"Herpes Zoster Oticus," etc.

His most recent publication was an article in the Pennsylvania Medical Journal for June, 1929, on Herpes Zoster in Otolaryngology.

He is survived by his wife and two sons.

Dr. Greenfield Sluder

Dr. Greenfield Sluder of St. Louis, a graduate of Washington University School of Medicine, St. Louis, 1888, died October 9, 1928, at Barnes Hospital, St. Louis, of pneumonia following amputation of the right leg, due to infection. Dr. Sluder was sixty-three.

Instead of the formal single year in the St. Louis City Hospital, the training school which has produced so many of our best general and special surgeons, Dr. Sluder had four. He could easily have practiced as a general surgeon when he left the hospital, but he elected instead to spend six years in general medicine with Dr. W. E. Fischel, then in the businest period of his distinguished career. Therefore when Dr. Sluder went to Europe for special training in rhinology and laryngology he was

already a fully developed physician and surgeon with a record of successful accomplishment. He was already a successful teacher, a function he prized to the end of his days because it multiplied his usefulness to his fellows and to his community.

His foreign training was exacting and laborious. Original work on the physiology and minute anatomy of the larynx brought to him the high regard of his instructors. He said he did not see a patient for ten months. His early work was all in the laboratory. When he returned to St. Louis to enter his specialty he had in mind not the mere acquisition of patients and the development of a lucrative practice. These came to him as a matter of course. He knew he had capacity to solve some of the most intricate problems in rhinology and laryngology, and he diligently pursued such anatomical and physiological studies as he knew would best equip him for the work. He always had a problem in process of solution, for he never rested. Even when on "vacation" at Harbor Point, he carried his books and manuscripts, often in the quiet of his den getting the opportunity to do thinking which his busy life at home forbade.

So the substantial advance he brought to us in knowledge of the tonsils, the neurology of the paranasal sinuses, the hyperplastic bone changes, the swallowing reflex, the vacuum headache (worked out with Dr. Ewing), and the extended influence of the nasal ganglion, were the product of a cultured mind disciplined to laborious and continuous effort. Sluder's neuralgia is now well known, as is also the change at times brought about in the optic discs by sinus disease.

His manual training led him to devise special instruments to carry out specific procedures. The Sluder tonsillotome is based on a keen anatomical observation, coupled with a trained mechanical sense. He early recognized the principles of the light reflex in searching the nose and throat for obscure lesions, so he practically reproduced sunlight at each of his work-tables by means of a specially devised arc lamp.

Only those who worked with him (and some patients) realized the enduring patience with which he pursued his search for lesions. It was not unusual for him to keep a patient all day, making and recording observations twenty or more times. He was equally relentless with himself in his pursuit of truth and did his work so painstakingly that the courage of his convictions

rested on a very solid foundation and never gave way when bombarded by criticism.

Latterly his restless zeal impelled him to a study of the sympathetic nervous system, about which little is known. His most vivid expressions of impatience over the illness which began in Boston two or more years ago were brought out because he could not pursue these studies he had mapped out and which he felt competently trained to follow. He was unperturbed when the Fellows of the Association could not at the time accept his opinions, for he never presented a hastily prepared paper just for the sake of appearing on the program, and he scorned those who did. When he read or published a paper it was based on an adequate number of carefully checked observations and was ready to be added to the textbooks of his subject.

Such was Dr. Sluder in the field of science, an honest and diligent observer who kept his mental feet solidly on the ground of general medicine and whose broad general training, special training, and common sense made him a leader amongst men of foremost talent. Such men are rare. We shall not soon see his like again, and though he has left us a glorious heritage, we regret that he was not spared to enjoy the tributes he so fairly earned, growing with the years.

Dr. Sluder was indefatigable in the pursuit of knowledge concerning his work. He was a frequent contributor to periodical literature, some fifty titles being listed in English and German journals. In addition to this, he was the author of three monographs: "Concerning Some Headaches and Eye Disorders of Nasal Origin," 1918; "Tonsillectomy by Means of the Mandible and a Guillotine, with a Review of Collateral Issues," 1923; "Nasal Neurology, Headaches and Eye Disorders," 1927.

Dr. Myles Standish

Dr. Myles Standish was born in Boston, Massachusetts, October 17, 1851, and died on June 26, 1928.

He was educated at Bowdoin College, where he took his A. B. in 1875, and his A. M. in 1878. He graduated from Harvard Medical School in 1879. He also had an honorary S. D. from Bowdoin College in 1910.

His earlier medical career included an internship at Carney Hospital in 1879-80, postgraduate work at the University of

Berlin in 1880-81, and a house surgeonship at the Massachusetts Eye and Ear Infirmary, 1881-83. He had been Assistant Surgeon in the Massachusetts General Hospital, the Boston City Hospital, and the Carney Hospital, 1892-1913, and Assistant Surgeon of Ophthalmology at the Massachusetts Eye and Ear Infirmary, 1888-92, while at the same institution he became Ophthalmic Surgeon, 1892-1913, and consulting surgeon, 1913. At the Harvard Medical School he was Assistant in Ophthalmology 1892-1901, Instructor, 1901-04, Assistant Professor of Ophthalmology, 1904-09, Williams Professor of Ophthalmology, 1909-14, and Emeritus Professor from 1914. He retired as Captain on the Ambulance M. V. M. in 1891 and he had been in the United States Medical Reserve.

Dr. Standish was a frequent contributor to medical journals. He was a member of the Massachusetts Medical Society, the American Medical Association, the New England Ophthalmological Society, the American Ophthalmological Society (of which he was President in 1912). He retired from practice some years before his death.

Dr. Lewis H. Taylor

Lewis H. Taylor, born July 29, 1850, at Taylorsville (now Washington's Crossing), Bucks County, Pennsylvania, died at Wilkes-Barre, Pennsylvania, November 5, 1928. He was the son of Samuel B. and Margaret H. (Baker) Taylor, of old Quaker stock, who were among the settlers of the present site of Tacony, Pennsylvania, in the latter part of the seventeenth century.

In 1877 Dr. Taylor entered the School of Medicine of the University of Pennsylvania, receiving the degree Doctor of Medicine in 1880, the subject of his graduating thesis being "The Miscroscope and the Busy Practitioner." That summer he took special work in ophthalmology and otolaryngology in Philadelphia, and later, 1883-84, studied in Vienna. From 1885 to 1894 he was medical inspector of the Pennsylvania State Board of Health. He was President of the Luzerne County Medical Society in 1885, of the Medical Society of the State of Pennsylvania in 1912, and was chairman of the Section on Ophthalmology of the American Medical Association in 1906. In addition, he was a member of the Lehigh Valley Medical Association, of the Philadelphia Pathological Society, of the Medical Club of Philadelphia, of the American Academy of Ophthalmology and Oto-

laryngology, of the American Ophthalmological Society, of the American Otological Society, and of the American College of Surgeons. At the time of his death he was Librarian of the Luzerne County Medical Society, having been founder of the library and its patron since 1893. He was the first resident physician to be appointed for duty at the Wilkes-Barre General Hospital, had been Ophthalmologist on the staff and President of the Board of Directors.

In 1891 he received the degree Master of Arts from Lafayette College.

Dr. Taylor married Emily Beard Hollenbeck of Wilkes-Barre, on June 4, 1884. The widow and one daughter survive.

Dr. Taylor was author of the following:

"Report of the Epidemic of Typhoid Fever at Plymouth, Pa." (1885)
"Typhoid Fever and Impure Milk" (1892)
"Typhoid Fever in Wilkes-Barre, Pa." (1889)
"The Medicinal Specialist" (1901)
"A Case of Foreign Body Driven Through the Tissue of the Nose Into the Orbit—Recovery" (1886)
"Ocular Troubles as Influenced by Nasal Disease" (1888)
"A Severe Case of Quinsy in a Child Eight Months Old" (1890)
"A Case of Brain Tumor with High Grade Choked Disc—Autopsy" (1891)
"Caries of the Wall of the Auditory Canal" (1891)
"Symmetrical Dislocation of Both Lenses Upward and Outward—Congenital" (1893)
"Conservative Treatment of Wounds of the Eyeball" (1894)
"Address in Otology" (1895)
"Shot Gun Wounds of the Eye" (1896)
"Mastoid Disease" (1899)
"The Operation of Couching Performed by a Mule" (1902)
"Treatment of Suppurating Ulcers of the Cornea" (1904)
"A Case of Steel in the Eyeball which the Haab Magnet Failed to Remove" (1906)
"Commencement Address, Wilkes-Barre City Hospital" (1923)
"Some Old Books and Their Owners" (1920)
"Our Library" (1925)
"Dr. William Beaumont" (1927)

The members, in memory of the departed, stood in silence for one minute.

UNFINISHED BUSINESS:

DR. W. H. WILDER: The Committee on Optics and Visual Physiology would like to have a vote of the Academy, an endorsement of the report and adoption of the recommendation in regard to measurements. Such an endorsement at this time will aid us very much in gaining the cooperation of the Bureau of Standards which we very much desire.

DR. W. B. LANCASTER: I move that the Academy approve of the suggestion of the Committee that vision be measured by the metric system. (Motion seconded by Dr. E. C. Ellett and carried).

BY-LAW CHANGES:

Proposal No. 1

It has been well known for some time that the scheme of carrying on the financial part of the Academy could be improved upon, although we seem to have a fair credit balance in our reserve. At the Detroit meeting, a committee was appointed to devise a financial scheme. This involved the following changes in the By-Laws, which are submitted at this time.

Article 4, *The Treasurer*. Strike out the entire old paragraph and substitute the following:

"He shall collect all money due the Academy and deposit same to the Academy account."

The method of withdrawing these funds for expenses or investment is detailed in the appended financial plan.

1. Single bank account where monies are collected.
2. All expenditures must be authorized by the Council before incurred, except that regular current expenses and small accounts originating at conventions may be paid and approved at the next Council meeting.
3. All bills to be paid by check with signature of both Secretary and Treasurer.
4. Monthly financial statement sent to the President, Treasurer, and members of the Council.
5. An annual audit by an expert Auditor.

Investments

1. The President, Treasurer, and Secretary to constitute the Investment Committee. This committee to be limited in its choice of investments to those securities which would be acceptable in trust accounts. (Note): (This would mean bonds of the Government, State and certain municipalities. Most trust companies

recognize certain bonds of large corporations and occasionally a first mortgage on real estate.)

2. All securities belonging to the Academy to be held by a suitable trust company, which will collect all interest and deposit it to the Academy account. No security to be sold or exchanged without the consent of the entire Investment Committee.

DR. W. H. WILDER: I move that the deliberations of the Finance Committee be a matter of Unfinished Business for next year. (Motion seconded and carried).

Proposal No. 2

Increase the number of active Fellows on the Council from three (3) to four (4), each to serve for a period of four (4) years instead of three (3) years.

DR. A. J. BEDELL: I move the adoption of Proposal No. 2. (Motion seconded and carried.)

Proposal No. 3

When a member is elected to Fellowship in the Academy with the certificate of one of the national examining boards, he will be classified in the official membership list of the Academy as practicing the specialty for which he holds the certificate, and shall not be classified as both ophthalmologist and an otolaryngologist until he shall have received the certificate of both Boards.

DR. W. H. WILDER: I move the adoption of Proposal No. 3. (Motion seconded).

DR. E. C. ELLETT: I move to amend the Proposal so that it will apply to all present members of the Academy as well as all future members. (Amendment seconded).

DR. T. E. CARMODY: If you list them that way there will be a number of members who will not be listed because they do not have their certificate.

(Vote on the amendment, lost. Vote on the original motion, carried by rising vote).

DR. W. P. WHERRY: Relative to Proposal No. 1, the conducting of the business of the Academy. The Academy is now so large that it is in the "big business" class. Changes in the method of conducting our finances were proposed last year in St. Louis, but it has seemed wise to the Council at this meeting that before definite changes are made that might perhaps seem proper at this time but in their operation may prove to be no

better than those now in use, and since it is recognized that doctors are not particularly good financiers, it would be well to authorize, and the Council has authorized, the employment of a competent auditor in Chicago, and has appointed a committee consisting of Dr. Wilder, Dr. Large and myself, to meet with this auditor and an officer of some trust company, and together work out a good business-like system or method of handling the finances of this society. Because of that action the Council has asked that the vote on Proposal No. 1 be held over until after this committee has had its meeting, worked out its program, and is ready to report. The Council also wishes that something be put in the program for next year authorizing a change of By-Laws to that effect, so that when it comes up for action next year it can be put into effect immediately. With that in view, Dr. Wilder will offer a motion.

DR. W. H. WILDER: I move that the Council be authorized by this society to revise its Constitution and By-Laws in accordance with the statement you have just heard, incorporating these different items on which you have voted, and in order to facilitate that, to give the Council authority to notify each of the members at least thirty days before the next meeting, so that when it comes up for vote at that time it can be settled. (Motion seconded by Dr. Spencer and unanimously carried by rising vote).

NEW BUSINESS:

Presession Volume

DR. W. P. WHERRY: I did not care to talk upon the Presession Volume because I may be prejudiced. This is my personal opinion and in no way represents the Council, or any of the committees, nor does it represent the thought of any organized group in this body. My personal opinion is first that the Presession Volume is an expense hardly warranted by the good that accrues therefrom and secondly it entails a tremendous amount of work. Much of the work that has been done on the Presession Volume this year has been gratis, and it would be unreasonable to assume that the task of preparing this volume next year can be put over on the basis of love. Furthermore, I feel that an author approaching a meeting such as this, with a paper that has been written months previously, and already published, presents the subject matter in a less interested way than if it had been more

recently prepared and was entirely new to his audience. I am intensely interested that the programs be of the utmost profit to those in attendance at the meetings, therefore, my conclusion is that from all viewpoints the presession volume is a needless expense, a liability rather than an asset, and that it retards, rather than increases, attendance and interest at the meeting.

DR. ALLEN GREENWOOD: I planned to come here this evening and be one of a very small minority that opposed the Presession Volume. I am glad to see that some of the otolaryngologists are with me.

I would like to have the President ask for a showing of hands of everyone who read the Presession Volume from cover to cover. There are a few, but less than half of those here.

I feel that in a society such as this a Presession Volume is inadvisable. I believe it lessens discussion. I also feel the discussion noted in the past two days did not in any way measure up to that which we have had formerly, and I think it is owing to the Presession Volume. I like to come to these meetings and have an author get up and present his subject freshly. I did not read the Presession Volume for that reason. I want to listen to what a man has to say without prejudice; then I am ready to get up and discuss it. I would much rather read the transactions than the Presession Volume.

DR. LUTHER C. PETER: It is strange that we can look at this subject with such very different eyes. My reaction to the Presession Volume is that when we are enabled to read these papers quietly, as I think most of us do, at least those in which we are interested, we can come to the society with better preparation in the matter of discussion than when we listen to a paper which may be well presented, or indifferently presented, and which we would like to discuss, but we are not able to express ourselves as we would like. With the paper before us we can look up our records in support of certain points which the author may make, or to correct some ideas which we think should be corrected in the presentation of the paper. I cannot feel that an author is stale on his subject because it requires infinitely more preparation to present a subject correctly in ten minutes than to read a paper which will probably cover twenty minutes. If an author can summarize his paper, as he should, in ten minutes, he will hold our attention even better than if he spent twenty minutes in a rambling way covering the ground.

The Section on Ophthalmology of the American Medical Association has been accustomed to a Presession Volume for some years, and I doubt very much whether very many members would vote to abandon it.

The question of sending letters and telegrams to get these papers in can also be considered. I am speaking from experience with our former methods. When we used the abstract system we were obliged to send telegrams and letter and more letters in order to get in the short abstracts. In other words, this part of the program is exactly the same as under the old abstract system.

As to the expense, there has been no definite calculation of the acutal expense of this Presession Volume. If it is systematically arranged the form can be set up and carried over, just as we did at the International Congress held in Washington some years ago, with a comparatively small added expense for the Transactions. The cuts must be the same, but I honestly believe that our actual expense so far as this volume is concerned would not exceed fifty cents per member when it is actually analyzed.

Dr. W. P. Wherry: For Dr. Peter's benefit let me say that we have already paid approximately $2100.00 on the Presession Volume and Transactions, of which $980.08 has been charged to the Presession Volume and the balance to the Transactions. The charge of $980.00 consisted entirely of stock, cover, and the things that enter into the Presession Volume; the balance charged against the Transactions covers set-up, proofing and changes incident to proofing, cuts, etc. The cost of mailing the Presession Volume was approximately $224.

The President: The members of the society must realize that the discussion of this topic could go on indefinitely. The Chair feels that both sides have presented their arguments clearly, and that the best way to settle it is for the members to express their wish one way or the other. If someone will make a motion either to discontinue or to continue the Presession Volume it will bring the question to a head.

Dr. Samuel R. Skillern: I move that we discontinue the Presession Volume. (Motion seconded, but lost).

Dr. Ross H. Skillern: I happened to be at the meeting of Council when this discussion took place. It seemed to be absolutely divided between the otalaryngologists on the one side and the oph-

thalmologists on the other. The otolaryngologists did not want the Presession Volume continued, and the ophthalmologists did. The ophthalmologists in the American Medical Association have had it right along with great success, I understand. As you may recall, some years ago we tried it for one year and that was the end of it. It seems to me that the otolaryngologists do not want it. It is perfectly all right for the ophthalmologists to have it, but why should they compel us to take it if we do not want it? I will therefore move that the ophthalmologists be permitted to have their Presession Volume, and that the otolaryngologists give it up.

THE PRESIDENT: The Chair with great regret rules the speaker out of order.

Election of Members

DR. W. P. WHERRY: The following new members have been approved by the Council:

Adkins, George Eli, Jackson Infirmary, Jackson, Miss., ALR.
Amano, Kageyas Wat, 502 Woodland Terrace, Philadelphia, Pa., ALR.
Anthony, Daniel Harbert, 1720 Exchange Bldg., Memphis, Tenn., Op.
Atkinson, Thomas Ernest, Coronado Bldg., Greeley, Colo., ALR.
Barkan, Otto, 490 Post St., San Francisco, California, Op.
Barnett, Saul E., 25 Parsons St., Detroit, Michigan, ALR.
Beideman, Joseph Ellsworth, 24-28 Montgomery Trust Arcade, Norristown, Pa., Op.
Bolotow, Nathan A., 224 Thayer St., Providence, R. I., ALR.
Brawner, Leon Edward, 157 Forrest Ave., N. E., Atlanta, Georgia, ALR.
Bredlau, Ernest A., 2753 West North Ave., Chicago, Ill., ALR.
Briant, Thomas Ernest, Colbeck Clinic Bldg., Division St., Welland, Ontario, Can., ALR.
Carter, John M., 1613 David Whitney Bldg., Detroit, Mich., ALR.
Cave, Linus S., 608 E. Genesee St., Syracuse, N. Y., ALR.
Childers, Robert Jefferson, 604 Park Ave., Plainfield, N. J., ALR.
Codd, Alphonso N., Paulsen Medical and Dental Bldg., Spokane, Wash., ALR.
Colbert, Robert Martin, 160 W. 87th St., New York, N. Y., ALR.

Collier, George Benjamin, 1317 Whitney Bldg., New Orleans, La., ALR.

Cone, Alfred Jared, 404 Laurel Bldg., Muscatine, Iowa, ALR.

Constans, George Maurice, Quain and Ramstead Clinic, Bismarck, N. D., Op.

Crane, Walter Raymond, 907 Chapman Bldg., Los Angeles, Cal., ALR.

Davidson, Morris, 210 W. 70th St., New York, N. Y., Op.

Dean, Arthur Clark, Wallace Blk., Crookston, Minn., ALR.

Dixon, Fred Willis, 1029 Rose Bldg., Cleveland, Ohio, ALR.

Dlugasch, Louis, 110 E. 36th St., New York, N. C., ALR.

Duncan, Robert Austin, 339 Amarillo Bldg., Amarillo, Texas, ALR.

Dyer, Benjamin G., Ames, Iowa, ALR.

Dyer, Wallace Curtis, 207 Cleveland Life Bldg., Evansville, Ind., ALR.

Fowlkes, John Winston, 30 West 59th St., New York, N. Y., ALR.

Gamble, Richard Cotter, 30 N. Michigan Ave., Chicago, Ill., Op.

Geiger, Arthur Henry, 1725, 25 E. Washington St., Chicago, ALR.

Gill, William Davis, 323 Medical Arts Bldg., San Antonio, Texas, OALR.

Goldstein, Isadore, 1060 Park Ave., New York, N. Y., Op.

Good, William Murray, 63 Center St., Waterbury, Conn., ALR.

Goode, Edward Allan, 239 Sanford Ave., Flushing, N. Y., ALR.

Gordon, Glenn G., Baker Oregon, ALR.

Gowen, Francis V., 2033 Pine St., Philadelphia, Pa., ALR.

Gowen, George Robert, 306 Baker Bldg., Walla Walla, Wash., ALR.

Greear, James Noah, 1740 M St., N. W., Washington, D. C., Op.

Gresser, Edward Bellamy, 39 Fifth Ave., New York, N. Y., Op.

Griffey Edward Waddy, 1625 Main Blvd., Houston, Texas, ALR.

Gunn, Milus Liddell, Smith Bldg., Harlan, Kentucky, ALR.

Guttman, Melvin Reese, 2551 N. Clark St., Chicago, Ill., ALR.

Haessler, F. Herbert, 120 E. Wisconsin Ave., Milwaukee, Wis., Op.

Hanson, William Ludwig, 318 Murphy Bldg., East St. Louis, Ill., ALR.

Haralson, Charles Herndon, 816 Medical Arts Bldg., Tulsa, Okla., Op.

Harris, Thomas Jefferson, 104 E. 40th St., New York, N. Y., ALR.
Harter, John Howard, 320 Stimson Bldg., Seattle, Wash., ALR.
Hobart, Carl, 626 Metropolitan Bldg., St. Louis, Mo., Op.
Hofling, Charles A., 707 Race St., Cincinnati, Ohio, Op.
Hoople, Gordon D., Medical Arts Bldg., Syracuse, N. Y., ALR.
Horn, Walter L., 1040 Park Ave., New York, N. Y., ALR.
Howe, Spencer S., Bellingham, Washington, ALR.
Hughes, Wendell Lochead, 20 E. 53rd St., New York, N. Y., Op.
Hunt, Westley Marshall, 33 E. 68th St., New York, N. Y., ALR.
Irvine, Robert Steele, 490 Post St., San Francisco, Calif., ALR.
Jarvis, DeForest Clinton, Quarry Bank Bldg., Barre, Vermont, ALR.
Jenkins, Mike Clemont, 223 S. Main St., Pratt, Kansas, ALR.
Josephson, Emanuel M., 993 Park Ave., New York, N. Y., ALR.
Keefe, William John, 110 East Sidney Ave., Mount Vernon, N. Y., ALR.
Knowlton, Charles Davison, 520 Commonwealth Ave., Boston, Mass., ALR.
Lamb, Harry Howe, American Bank Bldg., Davenport, Iowa, ALR.
LeWin, Thurber, 112 Linwood Ave., Buffalo, N. Y., Op.
Lingeman, Edward Laurel, 406 Pennway Bldg., Indianapolis, Ind., ALR.
McCullough, Clarence Joseph, 625-35 Washington Trust Bldg., Washington, Pa., ALR.
McDavitt, Bertha Schenck Stecker, Woodson Eye, Ear, Nose and Throat Hospital, Temple, Texas, ALR.
McGarvey, William Edward, 802 Jackson City Bank Bldg., Jackson, Michigan, ALR.
Marks, Julian Barnett, 402 First Nat'l Bank Bldg., Milwaukee, Wis., ALR.
Martin, Roy David, Hastings, Nebraska, ALR.
Mayerberg, Emil R., 307 Medical Arts Bldg., Wilmington, Del., ALR.
Menger, Louis Charles, 202 Weirfield St., Brooklyn, N. Y., ALR.
Miller, Joseph William, 305 East 17th St., New York, N. Y., ALR.
Motto, Michael Paul, 550 Rose Bldg., Cleveland, Ohio, Op.
Nash, C. Stewart, 26 South Goodman St., Rochester, N. Y., ALR.
Nemours, Paul Roland, 3852 Greer Ave., St. Louis, Mo., ALR.

Oast, Samuel P., 30 East 40th St., New York, N. Y., Op.
Orr, Adrian V. B., 525 Welsh St., Chester, Pa., ALR.
Pelz, Mort D., Missouri Bldg., St. Louis, Mo., ALR.
Price, John Beadle, 513 Swede St., Norristown, Pa., ALR.
Richardson, James Joseph, Cook Hospital and Clinic, Ft. Worth, Texas, ALR.
Robb, Mayo, 323 Ellis Bldg., Phoenix, Arizona, ALR.
Romonek, Philip L., Medical Arts Bldg., Omaha, Neb., ALR.
Rosebrough, Frank Henry, 301-04 Moore Bldg., San Antonio, Tex., Op.
Rosenbaum, Morris, 61 W. 88th St., New York, N. Y., Op.
Rosenberger, Harry Clifford, 1065 Rose Bldg., Cleveland, Ohio, ALR.
Rudolphy, Jay Besson, 1924 Pine St., Philadelphia, Pa., Op.
Ruedemann, Albert Darwin, Cleveland Clinic, Euclid and 93rd St., Cleveland, Ohio, Op.
Runk, Roxette Landis, Washington Sanitarium and Hospital, Takoma Park, Washington, D. C., ALR.
Samuels, Bernard, 57 West 57th St., New York, N. Y., Op.
Sanders, John Gillis, Park Bldg., Mobile, Ala., ALR.
Schwartz, Virgil Joseph, 507 Physicians and Surgeons Bldg., Minneapolis, Minn., ALR.
Shuster, Benjamin Harrison, 1717 Pine St., Philadelphia, Pa., ALR.
Sidwell, Clarence Edward, Longmont, Colorado, ALR.
Smith, Carroll L., Paulsen Medical and Dental Bldg., Spokane, Wash., OALR.
Smith, Ruric N., 1017 Medical Arts Bldg., Tulsa, Okla., ALR.
Spencer, Shelby Cruthirds, 605 Columbus Ave., Waco, Texas, Op.
Spiesman, Irwin Gabriel, 25 E. Washington St., Chicago, Ill., ALR.
Sutherland, Jacob Marion, 8, 257, General Motors Bldg., Detroit, Mich., ALR.
Tassman, Isaac S., 715 Medical Arts Bldg., Philadelphia, Pa., Op.
Theobald, Georgiana Dvorak, 1800 S. Ashland Ave., Chicago, Ill., Op.
Theobald, John Joseph, 1800 S. Ashland Ave., Chicago, Ill., ALR.
Warner, Robert Jay, 320 Doctors Bldg., Nashville, Tenn., Op.
Wessels, Andrew Benjamin, 1305 Medico-Dental Bldg., San Diego, Calif., ALR.
Wiggin, William Irving, 202 Central St., Lowell, Mass., ALR.

Wolf, Samuel, 55 E. Washington St., Chicago, Ill., Op.
Woodward, Fletcher Drummond, Box 162, University, Va., ALR.
Ziegelman, Edward Frank, 273 B St., San Mateo, Calif., ALR.
Adler, Francis Heed, 313 S. 17th St., Philadelphia, Pa., Op.
Aronson, Sidney S., 23 E. Ohio St., Indianapolis, Ind., ALR.
Baeseman, R. Winfield, 928 Clinton St., Philadelphia, Pa., ALR.
Cardwell, Edgar Parmele, 12 Central Ave., Newark, N. J., ALR.
Constantinople, Panagiotes Stavros, 1801 Eye St., N. W., Washington, D. C., ALR.
Ebert, Edwin C., Naval Dispensary, Washington, D. C., ALR.
Fowler, Robert Henry, 153 E. 62nd St., New York, N. Y., ALR.
Fraser, Robert C., 940 Military St., Port Huron, Mich., ALR.
Hedrick, Harold B., 910 Rialto Bldg., Kansas City, Mo., ALR.
King, George L., Jr., 537 E. Market St., Alliance, Ohio, ALR.
Koebbe, Ezra Baldwin, Columbus, Nebraska, ALR.
Odeneal, Thomas Helm, 234 Cabot St., Beverly, Mass., OALR.
Parramore, James Buchanan, 322 St. James Bldg., Jacksonville, Fla., ALR.
Peery, Vance Price, Kinston, N. C., ALR.
Pinkerton, Forrest Joy, 46 Young Bldg., Honolulu, Hawaii, OALR.
Potter, Allen B., 3725 S. Kingshighway, St. Louis, Mo., ALR.
Rucker, Charles Wilbur, 910 Donaldson Bldg., Minneapolis, Minn., Op.
Simon, Frederick C., 914 Arcade Bldg., St. Louis, Mo., ALR.
Weaver, John O., Shenandoah, Iowa, ALR.
Weymann, Morie Frederick, 2007 Wilshire Blvd., Los Angeles, Calif., Op.

On motion of Dr. Wherry, duly seconded, the above candidates were declared members.

Dr. Allen Greenwood: I move a rising vote of thanks to Dr. Charlton, head of the local committee, and to all those who aided him in giving us such a wonderful time in Atlantic City. (Motion seconded and unanimously carried.)

Election of Officers

Dr. E. C. Ellett: The Council submits the following nominations for officers for next year:

President	William H. Wilder	Chicago, Ill.
President-Elect	John F. Barnhill	Miami Beach, Fla.
First Vice-President	Frank R. Spencer	Boulder, Colo.
Second Vice-President	Fred A. Kiehle	Portland, Oregon
Third Vice-President	Robert Sonnenschien	Chicago, Ill.
Executive Secretary	W. P. Wherry	Omaha, Nebr.
Secretary for Ophthalmology	William L. Benedict	Rochester, Minn.
Secretary for Otolaryngology	John Myers	Kansas City, Mo.
Secretary for Instruction	Harry S. Gradle	Chicago, Ill.
Treasurer	Secord H. Large	Cleveland, Ohio
Members of Council:	Fred Stauffer	Salt Lake City, Utah
	John M. Wheeler	New York, N. Y.
	John J. Shea	Memphis, Tenn.
	J. L. McCool	San Francisco, Calif.
Member of Board for Ophthalmic Examinations	Sylvester J. Beach	Portland, Me.
Editor Transactions		

The position of Editor of the Transactions is hard to fill, and at the request of the Council, an editorial board, consisting of the Executive Secertary and three others, will carry on the work of the Editor until a survey can be made and the proper man selected to present to you as Editor of the Transactions.

You have just passed an amendment to the By-Laws providing for an extension of the time of service of members of the Council to four years instead of three, and electing an additional member. The Council has interpreted that to mean that the present members of the Council who were elected for three years will serve four years, so that no member from the Fellowship of the Academy will go off this year. Dr. Stauffer, who is the Senior member, will hold an additional year, as well as Dr. Wheeler and Dr. Shea, and the Council recommends as an additional member to serve for four years, Dr. McCool of San Francisco.

Dr. A. J. Bedell: I move that the officers whose names have been read be declared elected. (Motion seconded and carried.)

THE PRESIDENT: The Chair will appoint Dr. Burch to escort the President-Elect to the platform.

DR. JOHN F. BARNHILL (President-Elect): Mr. President and Members of the Academy: No event in all my life has affected me more profoundly than the action you have just taken. This, however, should excite no curiosity when we recall that the Academy is the largest association of its kind in the world—not only the largest, but probably it wields a greater influence than any other society in our chosen field of work. Therefore, to be elevated to the Presidency of a body such as this is to me almost an overwhelming event.

There are several things that come to my mind now, one or two of which I will mention. In the first place, I feel somewhat like one of Shakespeare's characters—I very greatly desire to hold fast to the old friendships which I have made, and to bind them to my heart with hoops of steel; and I further desire to increase my friendships with the younger men who are coming in and who have come in recently, because it is through them that we shall receive the enthusiasm of youth which we so much need always.

This election, coming as it does without any solicitation from anywhere in so far as I know, as is the custom of this society and which should be the custom of all societies, makes the honor doubly dear to me.

I have but one promise, and that is that during the year which I shall preside over you, and for all future time as in the past, I shall wish to advance the interests of the Academy in so far as it is within me.

Selection of Place of Meeting for 1930

DR. E. C. ELLETT: The Council recommends as the place of meeting for 1930, Chicago, Illinois.

DR. A. J. BEDELL: I move that the Academy meet in Chicago in 1930. (Motion seconded and unanimously carried).

On motion, the Business Session adjourned.

SECTION ON OTOLARYNGOLOGY

Friday Afternoon Program

The Friday afternoon session convened at two-ten, the President, Dr. Harris P. Mosher, presiding.

EXHIBITION OF NEW INSTRUMENTS

Dr. W. H. Lyman, for Dr. W. D. Black of St. Louis, presented a tonsil pillar retractor which permits more thorough inspection of the tonsillar fossae than has formerly been possible.

Dr. T. S. Blakesley of Kansas City, presented the new head lamp which he had presented to the Section on Ophthalmology.

Dr. Sidney Yankauer, New York City, presented a pharyngeal speculum and a probe used in connection with it—a stiff steel probe long enough to go through the instrument.

Dr. Joseph C. Beck of Chicago presented the instrument for removal of adenoids which he exhibited in 1928, advising that it had proved to be a failure.

Dr. William V. Mullin, Cleveland, Ohio, read a paper entitled "Salivary Glands—Diseases, Treatment." This paper was discussed by Drs. William Mithoefer, Cincinnati, Ohio; T. E. Carmody, Denver, Colorado; Lyle S. Powell, Lawrence, Kansas; Harris P. Mosher, Boston; and William V. Mullin.

Dr. Sidney Yankauer, New York City, read a paper entitled "Nasopharyngeal Abscess. A Report of 155 Cases." This paper was discussed by Drs. Joseph C. Beck, Chicago; Samuel Salinger, Chicago; and Sidney Yankauer.

SYMPOSIUM OF THE ETHMOID:

The following papers were presented in this symposium:

"The Surgical Anatomy of the Ethmoidal Labyrinth," Dr. H. P. Mosher, Boston, Mass.

"Pathology and Diagnosis," Dr. Ross Hall Skillern, Philadelphia, Pennsylvania.

"Conservative Treatment of Ethmoiditis," Dr. Samuel Iglauer, Cincinnati, Ohio.

"Radical Treatment of the Ethmoid—Intranasal," Dr. Ralph A. Fenton, Portland, Oregon.

"External Radical Approach—Ethmoidal Sinusitis," Dr. R. C. Lynch, New Orleans, La.

This symposium was discussed by Drs. Joseph C. Beck, Chicago; Eugene R. Lewis, Los Angeles, California; John S. Pratt, Minneapolis, Minnesota; S. Fineman, New York City; Burt R. Shurly, Detroit, Michigan; and Harris P. Mosher and Ralph A. Fenton.

The scientific session of the American Academy of Ophthalmology and Otolaryngology adjourned *sine die*.

ALPHABETIC LIST

Honorary Fellows

*Birkett, H. S.....1190 Mountain St., Montreal, Canada, ALR
Dundas-Grant, Sir James.....148 Harley St., W. 1, London, Eng.
Elliot, Lt.-Col. R. H.
54 Welbeck St., Cavendish Sq., W., London, Eng.
Elschnig, Prof. A., Ferdinandstrasse, 10, Prague, Czecho-Slovakia
Graves, Basil.....35 Weymouth St., W., London, Eng.
Gray, Albert A.....Glasgow, Scotland
Johnson, Lindsay, Britannia Bldg., West St., Durban, Natal, Africa
Kirkpatrick, Lt.-Col. Henry, 54 Welbeck St., W. 1, London, Eng.
Lawford, J. B.....27 Weymouth St., Portland Pl., W., London, Eng.
Lister, Sir William T.....24 Devonshire Place, W. 1, London, Eng.
Nagar, Prof. F.....Freiestrasse 20, Zurich, Switzerland
*Tilley, Herbert.....University College Hospital, London, Eng.
Van der Hoeve, Prof. J.....Rijnsburgerweg, 6 A, Leiden, Holland

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Life Members

Brown, Edward J. (1911),
900 Nicollet Ave., Minneapolis, Minn., OALR
Day, Ewing W.....121 University Place, Pittsburgh, Pa., OALR
Dayton, W. L.(1896).....302 Funke Bldg., Lincoln, Neb., OALR
*Foster, Hal(1894)..1014 Chambers Bldg., Kansas City, Mo., ALR
*Jackson, Edward(1902).....314 14th St., Denver, Colo., Op.
Lochard, L. B.....Metropolitan Bldg., Denver, Colo., OALR
Mayer, Emil(1905)
666 West End Ave., New York City, N. Y., ALR
Posey, Wm. C.....2049 Chestnut St., Philadelphia, Pa., Op.
Stewart, T. M.....901 Union Trust Bldg., Cincinnati, O., OALR
Wood, Casey A.....7 W. Madison St., Chicago, Ill., OALR
Woods, Hiram.....842 Park Ave., Baltimore, Md., Op.A

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* Attended 1929 meeting. Numbers in parenthesis indicate years in which the Fellow became a member of the Academy. Fellows are requested to supply missing data and report errors in addresses or specialties to the Executive Secretary, Dr. W. P. Wherry, Medical Arts Bldg., Omaha, Nebr.

Fellows

A

*ABBOTT, W. J.(1919).....8314 Euclid Ave., Cleveland, O., OALR
*ABRAHAMSON, IRA A.(1927)
605 Traction Bldg., Cincinnati, O., Op.
ADAMS, A. L.(1905)....Professional Bldg., Jacksonville, Ill., OALR
*ADAMS, C. F.(1908).....34 West State St., Trenton, N. J., OALR
*ADAMS, CHARLES JOSEPH(1928)
618 A. & L. Bldg., Kokomo, Ind., OALR
ADAMS, D. S...508 Hume-Mansur Boldg., Indianapolis, Ind., OALR
ADAMS, ELDRIDGE S.(1920), Moore Bldg., San Antonio, Tex., ALR
ADAMS, FRANK M.(1924),
122 Waterman St., Providence, R. I., ALR
ADAMS, FRED'K.(1920),
Roosevelt Clinic, 1114 Boylston Ave., Seattle, Wash., OALR
ADKINS, GEO. ELI(1929)..Jackson Infirmary, Jackson, Miss., ALR
*ADLER, FRANCIS HEED(1929),
313 S. 17th St., Philadelphia, Pa., Op.
AGNEW, F. F.(1920), 120 3rd Ave., S. E., Independence, Ia., OALR
AINSLIE, GEORGE(1921).....Selling Bldg., Portland, Ore., OALR
ALBRIGHT, LT.-COM. P. M., M. C. U. S. Navy,(1922)
U. S. Naval Academy, Annapolis, Md., OALR
*ALDEN, ARTHUR M.(1922), 537 Frisco Bldg., St. Louis, Mo., ALR
ALDERDYCE, WM.(1908).....513 Madison Ave., Toledo, O., OALR
ALEXANDER, GEO. J.....1629 Spruce St., Philadelphia, Pa., ALR
ALKIRE, H. L.(1898).....614 Kansas Ave., Topeka, Kan., OALR
ALLEN, CHAS. E.(1920)
712 Waldheim Bldg., Kansas City, Mo., OALR
ALLEN, G. HENRY(1916).....Mills Bldg., Topeka, Kan., OALR
*ALLEN, G. V.(1921).....835 Kansas Ave., Topeka, Kan., OALR
ALLEN, J. H.....Republic Bldg., Denver, Colo., ALR
*ALLEN, ORRIS T.(1928)
422 Rose Dispensary Bldg., Terre Haute, Ind., OALR
ALLEN, THOMAS DYER(1928)
122 S. Michigan Ave., Chicago, Ill., Op.
ALMOUR, R. W.(1925), 51 W. 73rd St., New York, N. Y., ALR
ALTER, F. W.....316 Michigan St., Toledo, O., OALR
ALTRINGER, A. N.(1920), 710 Rialto Bldg., Kansas City, Mo., ALR
ALVIS, BENNETT Y.(1928), 900 Carleton Bldg., St. Louis Mo., Op.
ALWAY, ROBERT D.....323 S. Lincoln St., Aberdeen, S. D., OALR
*AMANO, K. W.(1929), 502 Woodland Ter., Philadelphia, Pa., ALR
AMBERG, EMIL.....662 Maccabee Bldg., Detroit, Mich., ALR
AMBROSE, ELMER C.(1927).....910½ Main, Trenton, Mo., OALR
ANDERSON, CARL M.(1926)...Mayo Clinic, Rochester, Minn., ALR
ANDERSON, W. B.....Med. Arts Hosp., Brownwood, Tex., OALR
*ANDREWS, A. H.(1896)...30 N. Michigan Ave., Chicago, Ill., OALR

ANDREWS, B. F.(1912)..25 E. Washington St., Chicago, Ill., OALR
ANDREWS, H. D.(1912).....33 Allen St., Buffalo, N. Y., Op.
ANDREWS, M. P.(1915)
Manitowoc Sav. Bk. Bldg., Manitowoc, Wis., OALR
ANNON, W. T.....235 S. 15th St., Philadelphia, Pa., ALR
*ANTHONY, DANIEL HARBERT(1929)
Exchange Bldg., Memphis, Tenn., Op.
*APPLEMAN, L. F.(1913), 308 South 16th St., Philadelphia, Pa., Op.
ARBUCKLE, MILLARD F.,
1011 Beaumont Med. Bldg., St. Louis, Mo., ALR
ARMSTRONG, R. M.(1928)
1010 Fayette Bk. Bldg., Lexington, Ky., OALR
*ARNOLD, FRANCIS J.....182 Pearl St., Burlington, Vt., OALR
*ARONSON, SIDNEY S.(1929)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
ARRELL, W.....317 Main St. E., Hamilton, Ontario, OALR
ARROWSMITH, HUBERT, 11 Schermerhorn St., Brooklyn, N. Y., ALR
ASBURY, MARY K.(1925)...3549 Holly Lane, Cincinnati, O., Op.
ASH, GARRETT G.(1920).....1 Main St., Bradford, Pa., OALR
ASHLEY, T. W.....5711 8th Ave., Kenosha, Wis., OALR
ATKINSON, D. T.(1918)
827 Med. Arts Bldg., San Antonio, Tex., OALR
ATKINSON, THOMAS E.(1929), Coronado Bldg., Greeley, Colo., ALR
ATKINSON, WALTER S.(1925),
168 Sterling St., Watertown, N. Y., Op.
AUERBACH, JULIUS..1016 Fifth Ave., New York City, N. Y., ALR
AYNESWORTH, H. T.(1912)
1004 Professional Bldg., Waco, Tex., OALR

B

BABBITT, JAMES A.(1924), 1912 Spruce St., Philadelphia, Pa., ALR
BAESEMAN, R. WINFIELD(1929),
928 Clinton St., Philadelphia, Pr., ALR
*BAHN, CHAS. A.(1922),
1707 Pere Marquette Bldg., New Orleans, La., Op.
BAILEY, FRED W.(1912),
Merchants Nat'l Bk. Bldg., Cedar Rapids, Ia., OALR
BAILEY, HAROLD(1908), 840 Landers Bldg., Springfield, Mo., OALR
BAIRD, CHAS. G.(1920), 301 E. Chapel St., Santa Maria, Cal., OALR
BAKER, CHAS. H.....Bay City Bk. Bldg., Bay City, Mich., OALR
BAKER, HARRY B.(1916).....59 Main St., Taunton, Mass., OALR
*BALDWIN, KATE W.....1117 Spruce St., Philadelphia, Pa., ALR
*BALENTINE, P. L.(1915), 1524 Chestnut St., Philadelphia, Pa., Op.
*BALL, MICHAEL V.....316 Hazel St., Warren, Pa., OALR
BALLENGER, H. C.(1915), 25 E. Washington St., Chicago, Ill., ALR
BALLIN, M. J.....20 E. 76th St., New York City, N. Y., ALR
BALLON, D. H.(1924).....1471 Crescent St., Montreal, Can., ALR
BANE, WM. C.....1005 Republic Bldg., Denver, Colo., OALR

BANE, W. M.....1005 Republic Bldg., Denver, Colo., OALR
BARBER, F.(1913).....75 S. Fitzhugh St., Rochester, N. Y., Op.
*BARDENHEIER, F. G. A.(1909)...Frisco Bldg., St. Louis, Mo., ALR
BARKAN, ADOLPH.....490 Post St., San Francisco, Cal., OALR
BARKAN, H.....490 Post St., San Francisco, Cal., Op.
BARKAN, OTTO(1929).....490 Post St., San Francisco, Cal., Op.
BARKER, CHAS. B.(1920)
224½ W. Okla. Ave., Guthrie, Okla., OALR
*BARKER, O. G. A.(1909),
804 Johnstown Trust Co., Johnstown, Pa., Op.
BARLOW, ROY A.....Hiram Sibley Bldg., Rochester, N. Y., ALR
BARNES, L. E.(1914), 6857 South Halsted St., Chicago, Ill., OALR
BARNETT, SAUL E.(1929).....25 Parson St., Detroit, Mich., ALR
*BARNHILL, J. F.(1896),
5369 Pine Tree Drive, Miami Beach, Fla., ALR
BARTHOLOMEW, A. C.(1904),
1020 Harrison St., Ft. Wayne, Ind., OALR
*BASS, A. L.(1924).....738 Francis Bldg., Louisville, Ky., OALR
BAUM, H. L.(1917).....510 Republic Bldg., Denver, Colo., ALR
BEACH, B. S.(1927).....140 W. 86th St., New York, N. Y., Op.
*BEACH, S. J.(1914).....704 Congress St., Portland, Me., Op.
BEAL, HOMER A.(1926), 1002 Argyle Bldg., Kansas City, Mo., ALR
*BEARD, HAROLD JOLLY(1924),
510 Dollar Bank Bldg., Youngstown, O., OALR
BEATTIE, ROBERT(1906),
David Whitney Bldg., Detroit, Mich., OALR
BEATTIE, WILLIAM H.(1921), 258 Genesee St., Utica, N. Y., OALR
BEATTY, H. G.....681 E. Broad St., Columbus, O., ALR
BEAUDOUX, HARRY A.....230 Grand Ave., Oakland, Cal., OALR
*BECK, A. L.(1927)..421 Huguenot St., New Rochelle, N. Y., OALR
BECK, FREDERICK L.(1920),
408 Hynds Bldg., Cheyenne, Wyo., OALR
*BECK, JOS. C.(1901).....185 N. Wabash Ave., Chicago, Ill., ALR
BECKER, H. M.....49 S. 4th St., Sunbury, Pa., OALR
*BEDELL, ARTHUR J.....344 State St., Albany, N. Y., Op.
BEEBE, C. S. .....120 E. Wisconsin Ave., Milwaukee, Wis., Op.
BEEM, IONE F.(1916).....31 N. State St., Chicago, Ill., OALR
BEGLE, HOWELL LLEWLLYN(1928),
2730 E. Jefferson Ave., Detroit, Mich., Op.
*BEIDEMAN, J. E.(1929),
Montgomery Trust Arcade, Norristown, Pa., Op.
BEIL, J. W.(1918).....Argyle Bldg., Kansas City, Mo., OALR
BELL, M. H.(1913), First Nat'l Bank Bldg., Vicksburg, Miss., Op.
BELL, WILL OTTO(1928)
1431 Medical Dental Bldg., Seattle, Wash., Op.
BELLIN, JOSEPH.....523 W. 6th St., Los Angeles, Cal., OALR
BELLOWS, GEORGE E.(1896), Rialto Bldg., Kansas City, Mo., Op.
*BENEDICT, WILLIAM L.(1919), Mayo Clinic, Rochester, Minn., Op.
*BENNETT, ARTHUR G.....150 N. Pearl St., Buffalo, N. Y., Op.

BENTLEY, NEIL I., 1161 David Whitney Bldg., Detroit, Mich., OALR
*BERENS, CONRAD (1924), 30 E. 40th St., New York City, N. Y., Op.
BERNATZ, C. F. . . . . Park Bldg., Pittsburgh, Pa., Op.
BERNSTEIN, E. J. (1900) . . . . . 10 Peterboro, Detroit, Mich., OALR
BERRISFORD, PAUL D. . . . . Lowry Bldg., St. Paul, Minn., OALR
BERRY, D. F. (1911),
710 Hume-Mansur Bldg., Indianapolis, Ind., ALR
BETTISON, D. L. . . . . Medical Arts Bldg., Dallas, Tex., ALR
*BEYER, A. G. (1925) . . . . . 19 Garfield Pl., Cincinnati, O., OALR
*BEYER, LOUIS J. (1918) . . . . 449 Delaware Ave., Buffalo, N. Y., ALR
BINGER, H. E. (1917) . . Lowry Medical Bldg., St. Paul, Minn., OALR
BINKLEY, ROY C. (1915) . . . . . 530 Fidelity Bldg., Dayton, O., OALR
*BIRSNER, LOUIS J. (1926), 25 1st Nat'l Bk. Bldg., Belleville, Ill., ALR
*BLAAUW, E. E. . . . . 190 Ashland Ave., Buffalo, N. Y., Op.
BLACK, MELVILLE (1904),
424 Metropolitan Bldg., Denver, Colo., Op.
BLACK, NELSON MILES,
120 E. Wisconsin Ave., Milwaukee, Wis., Op.
BLACK, PAUL (1927) . . . . . Stuart Bldg., Lincoln, Neb., Op.
BLACK, W. D. (1900) . . . . Metropolitan Bldg., St. Louis, Mo., ALR
BLACKBURN, W. J. (1919) . . . . 663 Reibold Bldg., Dayton, O., OALR
*BLAIR, MORTIMER WARREN (1928)
N. E. cor. 20th and Chestnut Sts., Philadelphia, Pa., Op.
BLAIR, W. W. . . . . 121 University Place, Pittsburgh, Pa., Op.
BLAKE, WM. F. . . . . 490 Post St., San Francisco, Cal., Op.
*BLAKESLEY, T. S. . . . . Lathrop Bldg., Kansas City, Mo., OALR
BLASSINGAME, C. D. (1924)
414 Phys. and Surg. Bldg., Memphis, Tenn., ALR
BLEDSOE, R. W. (1908), 1005 Madison Ave., Covington, Ky., OALR
BLICKENSTAFF, A. J. (1922) . . . Peoria Life Bldg., Peoria, Ill., OALR
BLUE, J. B. (1916) . . . . . Columbian Tower, Memphis, Tenn., OALR
BLUM, HENRY N.,
1211 Maison Blanche Bldg., New Orleans, La., OALR
BOBB, E. V. (1918),
101 First Nat'l Bk. Bldg., Mitchell, S. D., OALR
BOECKMANN, EGIL . . . . . Medical Arts Bldg., St. Paul, Minn., OALR
BOEHRINGER, W. H.,
700 Central Medical Bldg., Philadelphia, Pa., OALR
BOERNER, MORRIS H. (1923) . . . Norwood Bldg., Austin, Tex., OALR
BOILER, W. F. (1912) . . . . . Dey Bldg., Iowa City, Ia., OALR
*BOLOTOW, NATHAN A. (1929)
108 Waterman St., Providence, R. I., ALR
*BONE, HERMAN D. . . . . 19 Pleasant St., Gardner, Mass., OALR
BONNER, WM. F. (1926)
224 Del. Tr. Bldg., Wilmington, Del., OALR
BOONE, J. C. (1921) . . . 213 Pythian Bldg., South Bend., Ind., ALR
BOONE, J. L. (1924) . . . . Professional Bldg., Jacksonville, Fla., ALR
BOOT, G. W. (1909) . . . . . 811 Sherman Ave., Evanston, Ill., OALR
BORDLEY, JAMES, JR. . . . . 330 N. Charles St., Baltimore, Md., OALR

BOUCHER, R. B.....414 Birks Bldg., Vancouver, B. C., OALR
BOURBON, OLIVER P.(1920),
607 S. Hill St., Los Angeles, Cal., OALR
BOUSQUET, J. P. E.(1924),
26 Sherbrooke St., W., Montreal, Can., OALR
BOUVY, HARRY M.(1920)....800 Pittock Blk., Portland, Ore., OALR
*BOUVY, L. B.(1927).....Foley Bldg., La Grande, Ore., OALR
BOYCE, G. H.(1912)....1st Natl. Bk., Iron Mountain, Mich., OALR
BOYCE, W. B.(1912)...1107 Ludington St., Escanaba, Mich., OALR
BOYD, E. T.(1896).....823 Majestic Bldg., Denver, Colo., OALR
BOYD-SNEE, HARRY....716 J. M. S. Bldg., South Bend, Ind., ALR
BOZER, HERRMANN EUGENE(1923)
468 Delaware Ave., Buffalo, N. Y., ALR
BRADFIELD, J. A. L....201 State Bank Bldg., La Crosse, Wis., OALR
BRASWELL, JAMES C.(1922)
1109 Medical Arts Bldg., Tulsa, Okla., OALR
BRAUNLIN, WM. H.(1920)
Marion Nat'l Bank Bldg., Marion, Ind., OALR
*BRAWLEY, F. E.....30 N. Michigan Blvd., Chicago, Ill., Op.
BRAWNER, LEON ED.(1929)
157 Forrest Ave., N. E., Atlanta, Ga., ALR
BRAY, E. R.(1912).....342 Lowry Bldg., St. Paul, Minn., OALR
BREDLAU, ERNEST A.(1929)
2753 W. North Ave., Chicago, Ill., ALR
BREHMER, HARRISON L.(1920)
1st Nat'l Bank Bldg., Albuquerque, N. M., OALR
BREWER, JOHN D.(1920).....Dyersburg, Tenn., OALR
*BRIANT, THOMAS E.(1929)
Colbeck Clinic Bldg., 23 Division St., Welland, Ont., ALR
*BRIBACH, EUGENE J.(1919)
212 Simpson Bldg., Atchison, Kan., OALR
*BRICKLEY, DANIEL W.(1918),
Uhler Phillips Bldg., Marion, O., OALR
BRIGGS, F. W.....Havre, Mont., OALR
*BRIGGS, H. H.....101 Haywood St., Asheville, N. C., OALR
BRIGGS, W. E.....1027 10th St., Sacramento, Cal., OALR
*BRITTEN, G. S.(1907)..Medical Arts Bldg., Syracuse, N. Y., ALR
*BROBST, C. H.(1907).....Central Bank Bldg., Peoria, Ill., OALR
BRODICK, FRANK W.....Central Trust Bldg., Sterling, Ill., OALR
BROOKS, EARL B.(1921).....939 Stuart Bldg., Lincoln, Neb., OALR
BROOKS, E. H.(1915)..108 W. College Ave., Appleton, Wis., OALR
BROSE, L. D.....501 South East First St., Evansville, Ind., OALR
BROWN, ALBERT LOUIS(1926),
2600 Union Central Bldg., Cincinnati, O., Op.
BROWN, A. O.(1927)....742 Maccabee Bldg., Detroit, Mich., OALR
*BROWN, C. M.....510 Delaware Ave., Buffalo, N. Y., ALR
BROWN, CHARLES WILLIAM(1927)
415 Elm St., San Diego, Cal., ALR
BROWN, CLYDE O.(1928), 201 Humboldt Bldg., St. Louis, Mo., ALR

*Brown, Edmund Towle.....30 Church St., Burlington, Vt., OALR
Brown, H. Alexander (1912)
291 Geary St., San Francisco, Cal., Op.
Brown, John C. (1920)
Medical Arts Bldg., St. Paul, Minn., OALR
Brown, John E. (1901)...370 East Town St., Columbus, O., OALR
Brown, J. M.....1136 W. 6th St., Los Angeles, Cal., ALR
Brown, L. E. (1917).....2nd Natl. Bank Bldg., Akron, O., OALR
Brown, Morrow D.....432 Republic Bldg., Denver, Colo., ALR
*Brown, Mortimer G. (1921)
604 Medical Arts Bldg., Syracuse, N. Y., ALR
*Brown, Samuel Horton (1910)
1930 Chestnut St., Philadelphia, Pa., Op.
Brown, W. Harold (1912)
Bk. of Montreal Bldg., Edmonton, Alberta, Can., OALR
Brownell, Morton Emmons (1928)
1019 First Nat'l Bank Bldg., Wichita, Kan., Op.
Bruder, Joseph (1908), 57 W. 57th St., New York City, N. Y., OALR
*Bruere, Gustave E. (1919)
Medical Dental Bldg., Portland, Ore., OALR
Brumm, Seth A. (1922)
Medical Arts Bldg., Philadelphia, Pa., OALR
*Bruner, Abram Breneman (1922)
1214 Guardian Bldg., Cleveland, O., OALR
Bruner, Wm. E.....Guardian Bldg., Cleveland, O., Op.
Brust, H. O. (1913)....820 S. Crouse Ave., Syracuse, N. Y., ALR
Bryan, James Leslie (1928)
919 Bennie-Dillon Bldg., Nashville, Tenn., OALR
Bryant, Alice G. (1908)....502 Beacon St., Boston, Mass., ALR
*Buchanan, Norman D. (1925)
479 Water St., Peterborough, Ont., Canada, OALR
Buckley, J. H. (1908)..1st Natl. Bk. Bldg., Ft. Smith, Ark., OALR
*Buckman, E. U. (1906), 70 S. Franklin St., Wilkes-Barre, Pa., OALR
Bulson, A. E. (1896)....406 W. Berry St., Ft. Wayne, Ind., OALR
Bulson, Eugene Loring (1928)
406 W. Berry St., Ft. Wayne, Ind., OALR
*Burch, Frank E. (1907)..754 Linwood Pl., St. Paul, Minn., Op.
Burdick, Austin F. (1925), 416 Tussing Blk., Lansing, Mich., OALR
Burke, John R. (1927).....207 Elm St., Holyoke, Mass., OALR
Burke, T. A.....Rose Bldg., Cleveland, O., OALR
Burleson, J. H. (1917)
Eye, Ear, Nose and Throat Hosp., San Antonio, Tex., OALR
Burns, S. S. (1919)..Missouri Theater Bldg., St. Louis, Mo., ALR
Burroughs, G. M. (1911).....5 Broad St., Danielson, Conn., Op.
Burton, Frank A. (1914)
Medico-Dental Bldg., San Diego, Cal., ALR
Bushman, L. B. (1927)
631 City Nat'l Bank Bldg., Omaha, Neb., OALR
*Butler, Margaret F....1831 Chestnut St., Philadelphia, Pa., ALR

BUTLER, ROBERT H.(1920)
112 W. Columbus Ave., Bellefontaine, O., OALR
BUTT, MIRIAM M.....2045 Chestnut St., Philadelphia, Pa., Op.
*BUVINGER, CHARLES W.(1921)
50 Washington St., East Orange, N. J., OALR
BYRNES, H. F.....67 Chesnut St., Springfield, Mass., OALR

C

*CAHILL, J. W.....390 Main St., Worcester, Mass., OALR
CALDWELL, ROBERT
831 New Donaghey Bldg., Little Rock, Ark., OALR
CALHOUN, F. PHINIZY(1911)
486 Peach Tree St., N. E., Atlanta, Ga., Op.
CALHOUN, JAS. G.....Metropolitan Bldg., St. Louis, Mo., Op.
CALLFAS, W. F.....1620 Med. Arts Bldg., Omaha, Neb., ALR
CAMERON, WM. G.....Puget Sd. Bk. Bldg., Tacoma, Wash., OALR
*CAMP, W. E.(1920)
Medical Arts Bldg., Minneapolis, Minn., OALR
CAMPBELL, C. H., Columbus State Bk. Bldg., Columbus, Neb., OALR
CAMPBELL, D. A.....David Whitney Bldg., Detroit, Mich. OALR
CAMPBELL, DON M.(1908)
David Whitney Bldg., Detroit, Mich., OALR
CAMPBELL, M. D.(1927)
David Whitney Bldg., Detroit, Mich., OALR
CAMPBELL, ROBERT A., 520 LaSalle Bldg., Minneapolis, Minn., OALR
*CAMPBELL, W. K.....96 Third Ave., Long Branch, N. J., OALR
CAPRON, F. P.....118 Angell St., Providence, R. I., OALR
CARDWELL, EDGAR P.(1929)..12 Central Ave., Newark, N. J., ALR
CARLETON, RALPH(1914)..10 Temple St., Springfield, Mass., Op.
CARMACK, J. W.(1919)
505 Hume-Mansur Bldg., Indianapolis, Ind., ALR
*CARMODY, T. E.(1904)....Metropolitan Bldg., Denver, Colo., ALR
CARMODY, THOMAS J.(1926)
408 The Temple Bldg., Danville, Ill., OALR
CARNCROSS, HELEN(1920)
58 E. Washington St., Chicago, Ill., Op.
CARPENTER, E. R.....Medical Arts Bldg., Dallas, Tex., OALR
*CARPENTER, E. W.(1920)....Prof. Bldg., Greenville, S. C., OALR
*CARR, GEO. W.(1909)..54 S. Franklin St., Wilkes-Barre, Pa., OALR
*CARROLL, GEO. G.....5 N. Goodman St., Rochester, N. Y., OALR
CARRUTH, HOWARD ERNEST(1928)
716 Med. Arts Bldg., Portland, Ore., OALR
*CARSON, W. E.....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
CARTER, JOHN M.(1929).David Whitney Bldg., Detroit, Mich., ALR
CARTER, W. W.(1896)..2 E. 54th St., New York City, N. Y., ALR
*CARVILL, LIZZIE MAUD(1914)
82 Commonwealth Ave., Boston, Mass., Op.
*CARY, E. H.....Medical Arts Bldg., Dallas, Tex., OALR

CARY, G. C.....Canon Block, Grand Junction, Colo., OALR
CASSADY, J. VERNAL(1928)
654 Associate Bldg., South Bend, Ind., OALR
CAVANAUGH, J. A.....1712-30 N. Mich. Blvd., Chicago, Ill., ALR
*CAVE, LINUS S.(1929)....Physicians Bldg., Syracuse, N. Y., ALR
CHADWICK, IRA B.(1920), 4 Terminal Bldg., Coffeyville, Kan., Op.
*CHAPMAN, S. JEFFERSON(1926)
402 Burns Bldg., Colorado Springs, Colo., ALR
CHAPMAN, V. A...110 E. Wisconsin Ave., Milwaukee, Wis., OALR
*CHAPPELL, R. S.(1925), 110 N. Illinois St., Indianapolis, Ind., ALR
CHARLES, JOS. W.....3720 Washington Ave., St. Louis, Mo., Op.
*CHARLTON, C. COULTER
124 S. Illinois Ave., Atlantic City, N. J., ALR
*CHASE, E. FRANK(1918)....1008 Cobb Bldg., Seattle, Wash., OALR
CHASE, S. B.(1925).....Carver Bldg., Ft. Dodge, Ia., OALR
CHASE, W. D.(1924).....230 E. Broad St., Bethlehem, Pa., ALR
CHATTIN, J. FRANKLIN.....671 Broad St., Newark, N. J., Op.
CHENERY, W. E.....377 Commonwealth Ave., Boston, Mass., ALR
CHENEY, JAMES W.....702 Schweiter Bldg., Wichita, Kan., OALR
*CHILDERS, ROBT. J.(1929)...604 Park Ave., Plainfield, N. J., ALR
CHILDS, H. A.....Eagle Blk., Creston, Ia., OALR
CHIPMAN, L. D. V.(1925)
42 Coburg St., St. John, N. B., Canada, OALR
CHRISTOPH, CARL H.(1928)
30 N. Michigan Ave., Chicago, Ill., OALR
*CLARK, H. S.(1916)
Medical Arts Bldg., Minneapolis, Minn., OALR
CLARK, I. G.(1920).....188 E. State St., Columbus, O., OALR
CLARK, J. SHELDON(1914), 509 State Bk. Bldg., Freeport, Ill., OALR
CLARK, L. H.(1915).....337 Monroe Ave., Rochester, N. Y., OALR
CLAY, G. E.(1917).....Med. Arts Bldg., Atlanta, Ga., Op.
CLEFF, OSCAR(1926).....55 E. Washington St., Chicago, Ill., OALR
CLEMENT, C. C.....30 N. Michigan Blvd., Chicago, Ill., OALR
CLERF, LOUIS HENRY(1923), 128 S. 10th St., Philadelphia, Pa., ALR
CLEVENGER, W. F.(1905)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
CLIFFORD, T. P.(1927)
1802 David Whitney Bldg., Detroit, Mich., OALR
CLOTHIER, JOSEPH.....Kane Bldg., Pocatello, Ida., OALR
*COATES, GEO. M.(1922).....1721 Pine St., Philadelphia, Pa., ALR
COBB, EDWIN (1912)
Masonic Temple Bldg., Marshalltown, Ia., OALR
*CODD, A. N.(1929)
Paulsen Medical and Dental Bldg., Spokane, Wash., ALR
*CODY, CLAUDE CARR(1928)
1304 Walker Ave., Houston, Tex., ALR
COFFELT, THEODORE A., 1240 Benton Ave., Springfield, Mo., OALR
COGAN, J. E.....707 Rose Bldg., Cleveland, O., OALR
*COHEN, HERMAN B.(1927)
Spruce & 13th St., Philadelphia, Pa., ALR

*COHEN, MARTIN....1 West 85th St., New York City, N. Y., Op.
*COHEN, SAMUEL(1920).....2009 Pine St., Philadelphia, Pa., ALR
COHN, FELIX.....47 W. 87th St., New York City, N. Y., ALR
COHN, SELDON.....Fulton, Ky., OALR
COLBERT, R. M.(1929)....160 W. 87th St., New York, N. Y., ALR
COLLIER, GEO. B.(1929)
1317 Whitney Bldg., New Orleans, La., ALR
COLLINS, E. W.....508 Majestic Bldg., Denver, Colo., ALR
COLLINS, J. D.(1914)....187 Main St., Northampton, Mass., ALR
COLVER, B. N.(1913).....Sanitarium 1, Battle Creek, Mich., ALR
COMEE, W. C.(1922)....Cady-Land Bldg., Green Bay, Wis., OALR
CONE, ALFRED J.(1929).....Laurel Bldg., Muscatine, Iowa, ALR
*CONLON, F. A.....301 Essex St., Lawrence, Mass., OALR
*CONNOR, CHAS. E.(1920).....Hamm Bldg., St. Paul, Minn., ALR
*CONNOR, RAY(1905).....1410 Stroh Bldg., Detroit, Mich., OALR
CONSTANS, GEO. M.(1929)
Quain and Ramstad Clinic, Bismarck, No. Dak., Op.
*CONSTANTINOPLE, P. S.(1929)
1801 Eye St., N. W., Washington, D. C., ALR
COOK, W. ALBERT(1924)..Medical Arts Bldg., Tulsa, Okla., OALR
COOPER, C. E.....652 Metropolitan Bldg., Denver, Colo., ALR
*COOVER, D. H.(1918), 820 Metropolitan Bldg., Denver, Colo., Op.
COPPS, LYMAN ALDEN(1923)
Marshfield Clinic, Marshfield, Wis., OALR
CORBETT, J. J.(1925).....520 Beacon St., Boston, Mass., OALR
*CORSER, J. B.....345 Wyoming Ave., Scanton, Pa., Op.
*COSTEN, J. B.(1925)
Beaumont Medical Bldg., St. Louis, Mo., ALR
COTT, C. C.....483 Delaware Ave., Buffalo, N. Y., ALR
COTT, D. N.(1924)
141 St. Marks Pl., St. George, Staten Isl., New York, ALR
COULTER, CHAS. F.
404 First Natl. Bk. Bldg., Great Falls, Mont., OALR
*COWAN, ALFRED(1928), 1930 Chestnut St., Philadelphia, Pa., Op.
*COWLEY, ROBERT HENRY(1926).....Berea, Ky., OALR
*COWPER, H. W.....543 Franklin St., Buffalo, N. Y., Op.
CRAFT, KENNETH L.(1920)
1003 Hume-Mansur Bldg., Indianapolis, Ind., ALR
*CRANE, J. WELLINGTON(1921)
128 Third Ave., Trenton, N. J., OALR
CRANE, W. R.(1929), 756 S. Broadway, Los Angeles, Cal., ALR
CREIGHTON, WM. J.(1910), 258 S. 18th St., Philadelphia, Pa., Op.
*CRISP, WM. H.(1913)....Metropolitan Bldg., Denver, Colo., Op.
*CROCKETT, R. L.....302 Main St., Oneida, N. Y., Op.
*CROMAN, JOS. M., JR.(1924)
Washington Apts., Mount Clemens, Mich., OALR
*CROSKEY, J. W.(1905)....Med. Arts Bldg., Philadelphia, Pa., Op.
*CROSS, A. E.(1914).....390 Main St., Worcester, Mass., Op.

*Cross, G. H.(1919) . . . . . .525 Welsh St., Chester, Pa., Op.
Crossley, E. R. . . . . .30 N. Michigan Blvd., Chicago, Ill., OALR
Crumrine, Norman Ross(1928), 595 Third St., Beaver, Pa., OALR
Culbertson, L. R.(1898) . . . .Masonic Temple, Zanesville, O., OALR
*Cullom, M. M.(1910)
1119 Bennie-Dillon Bldg., Nashville, Tenn., OALR
*Cunning, Daniel S.(1928)
114 E. 54th St., New York City, N. Y., OALR
Cunningham, Allan R.(1924)
260 Barrington St., Halifax, N. S., OALR
Curdy, R. J. . . . . .1134 Rialto Bldg., Kansas City, Mo., Op.
Curran, E. J.(1919)
1805 Federal Reserve Bk. Bldg., Kansas City, Mo., Op.
*Curry, Glendon E.(1912)
Westinghouse Bldg., Pittsburgh, Pa., Op.
*Curtin, Eugene A.(1923), 703 Connell Bldg., Scranton, Pa., OALR
Curtis, W. L. . . . . .702 Sharp Bldg., Lincoln, Neb., OALR
*Cushman, Beulah(1924)
25 E. Washington St., Chicago, Ill., Op.
Cutler, Franklin E. . . . .1025 Schofield Bldg., Cleveland, O., ALR

D

Dabney, S. G. . . . . .Starks Bldg., Louisville, Ky., OALR
Dabney, Virginius(1913)
1633 Connecticut Ave., Washington, D. C., ALR
Dailey, Gilbert L. . . . . .618 N. 3rd St., Harrisburg, Pa., OALR
Daily, Louis(1926) . .1117 Med. Arts Bldg., Houston, Tex., ALR
*Daily, Ray K.(1926) .1117 Med. Arts Bldg., Houston, Tex., OALR
Dancy, Alexander B.
First Natl. Bank Bldg., Jackson, Tenn., OALR
Daniels, R. L. . . . . .Elk's Bldg., New Bern, N. C., OALR
Darmer, Geo. A.(1921) . . . .401 Terminal Bldg., Aurora, Ill., OALR
Darrow, C. H.(1928), 110 Metropolitan Bldg., Denver, Colo., ALR
Davidson, H. P.(1924) . .58 E. Washington St., Chicago, Ill., Op.
*Davidson, Morris(1929) .210 W. 70th St., New York, N. Y., Op.
Davies, J. S.(1927) . . . . . .Washington Bldg., Tacoma, Wash., OALR
*Davies, T. A.(1913) . . . . . .578 Sherborne St., Toronto, Can., OALR
Davis, A. Edward. . . .40 E. 61st St., New York City, N. Y., Op.
Davis, Charles L.(1928)
3720 Washington Blvd., St. Louis, Mo., ALR
Davis, F. A.(1927), 224 W. Washington Ave., Madison, Wis., OALR
Davis, G. E. . . . . .1060 Fifth Ave., New York City, N. Y., OALR
Davis, Harry B.(1920), 712 Bryant Bldg., Kansas City, Mo., OALR
*Davis, J. Calvin, Jr.(1928), 425 Aquila Court, Omaha, Neb., OALR
Davis, J. E. . . . . .Kress Bldg., McAlester, Okla., OALR
Davis, J. L. . . . . .135 S. 18th St., Philadelphia, Pa., ALR
Davis, Ralph F.(1920) . . . . . .Mayer Bldg., Portland, Ore., OALR

*DAVIS, THOMAS W.
210 O'Hanlon Bldg., Winston-Salem, N. C., OALR
*DAVIS, W. T.(1919)...927 Farragut Sq., Washington, D. C., Op.
DAYTON, G. O.....1401 S. Hope, Los Angeles, Cal., OALR
.DEAL, JOHN FRANCIS(1928)
1st Natl. Bk. Bldg., Springfield, Ill., OALR
*DEAN, ALFRED, 308 Medical Arts Bldg., Grand Rapids, Mich., Op.
DEAN, ARTHUR C.(1929), Hot Springs Clinic, Hot Springs, S. D.,
DEAN, F. W.(1904).....532 First Ave., Council Bluffs, Ia., OALR
*DEAN, L. W.
Medical School, Washington University, St. Louis, Mo., ALR
DEAN, WALTER(1917)....300 Francis Bldg., Louisville, Ky., OALR
*DEBOE, M. P.....1st Natl. Bk. Bldg., Miami, Fla., OALR
DECHERD, H. B.....1515 Med. Arts Bldg., Dallas, Tex., OALR
DECKER, J. C.(1918).....Francis Bldg., Sioux City, Ia., OALR
DEFNET, WM. A.(1921)....1309 Stroh Bldg., Detroit, Mich., ALR
DEICHLER, LYN W.....1737 Chestnut St., Philadelphia, Pa., OALR
DELPH, JOHN FRANCIS(1928)
104 S. Michigan Blvd., Chicago, Ill., ALR
DENMAN, IRA O.(1915)....330 Richardson Bldg., Toledo, O., OALR
DENNIS, DAVID N.(1902).....221 W. Ninth St., Erie, Pa., Op.
DENNIS, EDWARD P.(1921).....309 W. 10th St., Erie, Pa., OALR
DENNIS, F. L.(1906)
301 Ferguson Bldg., Colorado Springs, Colo., ALR
DENNIS, GEO. J.(1910)...25 E. Washington St., Chicago, Ill., ALR
DERBY, GEO. S.....5 Bay State Rd., Boston, Mass., Op.
DERIVER, J. P.(1919), Veterans Bureau, San Francisco, Cal., OALR
DETLING, FRANK E.....500 S. Lucas St., Los Angeles, Ca., OALR
DEWEESE, CLARENCE..201 West Second St., Lexington, Ky., OALR
DEWEY, C. H.(1912), Maj. M. C., U. S. Army,
U. S. Army Hosp., Ft. Beni. Harrison, Indianapolis, Ind., OALR
DEWITT, EDWARD NICHOLAS(1928)
836 Myrtle Ave., Bridgeport, Conn., Op.A.
DICKSON, JOHN FORREST(1928)
418 Mayer Bldg., Portland, Ore., OALR
*DINTENFASS, HENRY.....1305 Spruce St., Philadelphia, Pa., ALR
DIXON, FRED W.(1929).....Rose Bldg., Cleveland, Ohio, ALR
DIXON, G. S.....218 2nd Ave., New York City, N. Y., R
DIXON, OTTO JASON(1928)
902 Med. Arts Bldg., Kansas City, Mo., ALR
DIXON, W. E....503 Med. Arts Bldg., Oklahoma City, Okla., OALR
DLUGASCH, LOUIS(1929)..110 E. 36th St., New York, N. Y., ALR
DOANE, L. L.(1905).....357 N. Main St., Butler, Pa., OALR
DODD, OSCAR(1903).....636 Church St., Evanston, Ill., Op.
*DOHERTY, WM. B.(1927)
150 W. 55th St., New York City, N. Y., Op.
DOLMAN, PERCIVAL(1919), 490 Post St., San Francisco, Cal.,Op.
DONNELL, N. R.(1917)...Metropolitan Bldg., St. Louis, Mo., Op.
DONOHER, WM. D.....Boston Bldg., Salt Lake City, Utah, OALR

DONOVAN, JNO. A.(1902)...507 Phoenix Bldg., Butte, Mont., OALR
DORENTE, D. R.....1st Natl. Bank Bldg., Fort Smith, Ark., OALR
DORGE, RICHARD IRVING(1922)
2319 Sixth St., South, Minneapolis, Minn., OALR
DOUGLAS, FREDERICK ALEXANDER(1922)
303 State Bank Bldg., La Crosse, Wis., OALR
DOW, FRANK E.(1914).....4 West St., Northampton, Mass., Op.
DOWLING, J. IVIMEY, 116 Washington Ave., Albany, N. Y., OALR
DOWLING, J. T.(1915)
Virginia Mason Hosp. Bldg., Seattle, Wash., OALR
*DUANE, J. F.(1916).....Jefferson Bldg., Peoria, Ill., OALR
DULANEY, OCTAVUS.....811 Brown Bldg., Louisville, Ky., ALR
DUNCAN, R. A.(1929)..339 Amarillo Bldg., Amarillo, Texas, ALR
DUNLAP, LAWRENCE G.(1920)
101 Main St., Anaconda, Mont., OALR
DUNN, MURISON.....Richmond, Ky., OALR
DUNN, O. B.....4th and Railroad Sts., Ironton, O., OALR
*DUNNINGTON, J. H.(1924)
30 W. 59th St., New York City, N. Y., Op.
DUNTLEY, G. S.(1913).....103 So. Side Sq., Macomb, Ill., OALR
DURR, S. A.(1927)
1304 Medico-Dental Bldg., San Diego, Cal., Op.
DUTROW, H. V.(1914)..1040 Fidelity Med. Bldg., Dayton, O., OALR
DYER, B. G.(1929).....Ames, Iowa, ALR
DYER, C. P.(1920).....408 Humboldt Bldg., St. Louis, Mo., Op.
DYER, WALLACE C.(1929)
206 Cleveland Life Bldg., Evansville, Ind., ALR

E

EAREL, A. M.(1912).....Willdon Bldg., Hoopeston, Ill., OALR
EARLEY, JAMES HUGH(1928)
1801 Eye St., N. W., Washington, D. C., ALR
EASTON, J. C.(1904), First Nat'l Bank Bldg., Springfield, O., OALR
EBER, CARL THEODORE(1928)
1006 Carleton Bldg., St. Louis, Mo., Op.
*EBERT, EDWIN C.(1929)
Naval Dispensary, Washington, D. C., ALR
EDSON, R. A.....498 Delaware Ave., Buffalo, N. Y., Op.
EDWARDS, C. J.(1913)
First National Bank Bldg., Vicksburg, Miss., OALR
EDWARDS, SLOCOMB R.(1918)
Christman Bldg., South Bend, Ind., OALR
*EGAN, BURTON WRIGHT(1928)
408 North St., Logansport, Ind., OALR
EIMER, CHARLES EUGENE(1928)
316 Lister Bldg., St. Louis, Mo., ALR
*ELLETT, E. C.(1898).....Exchange Bldg., Memphis, Tenn., Op.
ELLIS, CHARLES WARD(1928)..Bauch Bldg., Lansing, Mich., ALR

ELLIS, Z. H.(1927)..30 W. 59th St., New York City, N. Y., Op.
ELWOOD, CALVIN R.(1922)..Spies Bldg., Menominee, Mich., OALR
EMBERY, FRANK(1927)
1532 Overington St., Philadelphia, Pa., ALR
*EMERSON, LINN(1906)....Metropolitan Bldg., Orange, N. L., OALR
ENGLE, H. P.....Maytag Bldg., Newton, Ia., OALR
*ERSNER, MATTHEW S.....1915 Spruce St., Philadelphia, Pa., ALR
ESTABROOK, CHAS. T.....857 Pleasant St., Worcester, Mass., OALR
*EUBANK, AMBROSE E.(1928)
636 Argyle Bldg., Kansas City, Mo., OALR
*EVANS, J. N.(1925)..23 Schermerhorn St., Brooklyn, N. Y., Op.
EZELL, H. C.(1926)....228 Doctors Bldg., Nashville, Tenn., OALR

F

FAIRBAIN, JOHN F.....503 Delaware Ave., Buffalo, N. Y., ALR
FAIRCHILD, NORA M.(1922)..Med. Arts Bldg., Omaha, Neb., Op.
*FAIRING, JOHN W.(1924)
206 Tribune Review Bldg., Greensburg, Pa., OALR
*FAITH, THOMAS.....31 N. State St., Chicago, Ill., OALR
FALLOWS, HOWARD D.(1916)
.. Park Hospital Clinic, Mason City, Ia., OALR
*FARMER, A. G.(1919)....Fidelty Medical Bldg., Dayton, O., OALR
FARNSLER, H. HERSHEY..1438 Market St., Harrisburg, Pa., OALR
FARNSWORTH, M. A.(1917)
Crestone Heights Sanitarium, Colorado Springs, Colo., OALR
FARRELL, P. J. H.(1902), 25 E. Washington St., Chicago, Ill., OALR
FARRINGTON, P. M.(1904)
Physicians and Surgeons Bldg., Memphis, Tenn., ALR
FARRIOR, J. BROWN(1928)
605 Citizens Bank Bldg., Tampa, Fla., OALR
*FAWCETT, I.(1913).....75 12th St., Wheeling, W. Va., OALR
*FELT, C. L.....Chestnut & 20th Sts., Philadelphia, Pa., ALR
*FENTON, RALPH A.(1921)
806 Medical Arts Bldg., Portland, Ore., OALR
FERGUSON, E. S.(1906)
902 Medical Arts Bldg., Oklahoma City, Okla., OALR
*FERGUSON, R. J.(1927)....59 College St., New Haven, Conn., OALR
*FERMAN, JOHN W.(1921)....Stahlman Bldg., Charleroi, Pa., OALR
FERNLUND, GEORGE BYRON(1928)
Hillside Ave. and 118th St., Richmond Hill, New York, ALR
FERRIS, DAVID P.(1928), 1000 Carleton Bldg., St. Louis, Mo., ALR
*FIELDING, F. G.(1925).....70 Ridge St., Glen Falls, N. Y., ALR
FIGI, F. A.(1927).....Mayo Clinic, Rochester, Minn., LR
FINK, WALTER H.(1927)
426 LaSalle Bldg., Minneapolis, Minn., OALR
FINNOFF, W. C.(1916)....217 Imperial Bldg., Denver, Colo., Op.
FISCHER, N. A.(1925), 623 Union Trust Bldg., Pittsburgh, Pa., ALR
FISCHER, V. B.....203 Mercantile Bk. Bldg., Boulder, Colo., OALR

FISHER, CARL.....1930 Wilshire Blvd., Los Angeles, Cal., Op.
FISHER, JAMES A.(1925)
Jersey Central Bldg., Asbury Park, N. J., OALR
FISHER, W. A.(1896), 231 W. Washington St., Chicago, Ill., OALR
FISKE, GEO. F.....25 E. Washington St., Chicago, Ill., OALR
FITZGERALD, J. P.(1924), 104 S. Michigan Blvd., Chicago, Ill., Op.
FLAGG, J. D.(1900).....473 Virginia St., Buffalo, N. Y., Op.A.
FLATLEY, T. J.(1914).....Reliance Bldg., Moline, Ill., OALR
FLOYD, T. W.(1918).....811 Peoria Life Bldg., Peoria, Ill., OALR
*FLYNN, CHAS. T.(1927)
41 Thumbull St., New Haven, Conn., ALR
*FLYNN, JAMES A.(1924)
1511 Rhode Island Ave., N. W., Washington, D. C., OALR
*FOGARTY, CHAS. W.(1920)
St. Paul Clinic Bldg., St. Peter & 5th St., St. Paul, Minn., OALR
FOLEY, J. W.(1915).....Majestic Bldg., Denver, Colo., OALR
FOLK, MAX LYON(1928), 55 E. Washington St., Chicago, Ill., OALR
FOLKEN, FRANK GARRETT(1928)
306 Albert Lea State Bank Bldg., Albert Lea, Minn., OALR
FOLSOM, W. H.(1927)..11 N. Main St., Fond du Lac, Wis., OALR
*FORBES, SHERMAN B.(1921)
409 Citizens Bank Bldg., Tampa, Fla., OALR
FORD, HANBY LEWIS(1928)
401 Robeson Bldg., Champaign, Ill., OALR
*FORGRAVE, L. ROBERT(1922), 204 Logan Bldg., St. Joseph, Mo., OALR
FORSYTHE, S. T.(1919).....600 Rose Bldg., Cleveland, O., OALR
FORT, A. G.....303 Medical Arts Bldg., Atlanta, Ga., OALR
FOSTER, J. H.....1304 Walker Ave., Houston, Tex., ALR
*FOUTS, JOHN D.(1920).....910 Fidelity Bldg., Dayton, O., OALR
FOWLER, EARL B.(1925)..55 E. Washington St., Chicago, Ill., Op.
*FOWLER, ROBT. H.(1929)..153 E. 62nd St., New York, N. Y., ALR
FOWLER, S. R.(1910).....University Blk., Syracuse, N. Y., ALR
*FOWLER, WM.(1925)...1048 Maccabee Bldg., Detroit, Mich., OALR
FOWLKES, JOHN W.....30 W. 59th St., New York, N. Y., ALR
FOX, C. H.(1920).....2119 Central Ave., Kearney, Neb., OALR
*FOX, L. WEBSTER.....303 S. 17th St., Philadelphia, Pa., Op.
FOX, NOAH(1926).....841 E. 63rd St., Chicago, Ill., ALR
FOX, RALPH D.(1916).....Unity Bldg., Bloomington, Ill., OALR
FRANCHERE, FRED'K E.(1920)
Farmer's Loan and Trust Bldg., Sioux City, Ia., OALR
FRANK, IRA(1908).....104 S. Michigan Blvd., Chicago, Ill., ALR
FRANKEL, EDWARD, JR.(1928)
152 W. 58th St., New York City, N. Y., ALR
FRASER, R. H.(1925)
Battle Creek Sanitarium, Battle Creek, Mich., ALR
*FRASER, ROBT. C.(1929)..940 Military St., Port Huron, Mich., ALR
*FRASER, W. G.(1927)..Medical Arts Bldg., Ottawa, Canada, OALR
*FREEDMAN, L. M.(1920)
475 Commonwealth Ave., Boston, Mass., ALR

FREIMUTH, L. E.(1927)
234 University Club Bldg., St. Louis, Mo., ALR
FRENCH, R. F.(1913)...Masonic Temple, Marshalltown, Ia., OALR
FREUDENTHAL, WOLFF, 24 W. 88th St., New York City, N. Y., ALR
FRIEDENWALD, HARRY.....1212 Eutaw Pl., Baltimore, Md., Op.A.
FRIEDENWALD, J. S.(1925)..1212 Eutaw Pl., Baltimore, Md., Op.
FRINGER, W. R.....William Brown Bldg., Rockford, Ill., Op.
FROST, ALBERT DALBEY(1928)
681 E. Broad St., Columbus, O., Op.
FROTHINGHAM, EDWARD(1928)
8644 94th St., Woodhaven, N. Y., OALR
FUCHS, VALENTINE HENRY(1928)
220 Chaille Bldg., New Orleans, La., ALR
FULKERSON, C. B.(1914)
308 Kalamazoo Nat'l Bank Bldg., Kalamazoo, Mich., OALR
FULLENWIDER, C. M.....Barnes Bldg., Muskogee, Okla., OALR
FULLER, HARRY B.(1925)
231 W. Washington St., Chicago, Ill., OALR
FULLER, JOHN ANDRE(1928)
Farmers and Merchants Bank Bldg., Reno, Nev., OALR
*FULLER, T. E.(1915)
Texarkana Natl. Bank Bldg., Texarkana, Tex., OALR
FULTON, JOHN F.(1920)..833 Lowry Bldg., St. Paul, Minn., OALR
FURGASON, A. P.....Richards Blk., Lincoln, Neb., OALR
FURSTENBERG, A. C.(1925)
First National Bank Bldg., Ann Arbor, Mich., ALR
*FUST, JOHN H. E.(1922).....221 W. 9th St., Erie, Pa., OALR

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*GADBOIS, ARTHUR E.(1921), 421 Norfolk Ave., Norfolk, Neb., OALR
*GAILEY, WATSON W.(1923)
Griesheim Bldg., Bloomington, Ill., OALR
GALE, M. JEAN(1919).....Republic Bldg., Denver, Colo., Op.
*GALLAGHER, J. H. C.(1927), 1537 Main St., Springfield, Mass., ALR
GALLAHER, T. J.....California Bldg., Denver, Colo., ALR
GALLOWAY, T. C.(1927).....636 Church St., Evanston, Ill., ALR
*GAMBLE, R. C.(1929)....30 N. Michigan Ave., Chicago, Ill., Op.
*GARBER, J. M.....14 N. Mulberry St., Mansfield, O., OALR
*GARRAGHAN, EDW. F.....25 E. Washington St., Chicago, Ill., OALR
GARRETT, J. R.(1913).....Mt. Trust Bldg., Roanoke, Va., OALR
GATEWOOD, E. T.(1925)..Professional Bldg., Richmond, Va., ALR
GATEWOOD, W. L.....330 Park Ave., New York City, N. Y., ALR
GAUDET, LUCIEN SIDNEY(1928)
405 Franklin St., Natchez, Miss., OALR
GEIGER, A. H.(1929), 1725:25 E. Washington St., Chicago, Ill., ALR
GEIGER, C. W.(1904).....Arcade Bldg., Kankakee, Ill., OALR
*GEORGE, HENRY W.(1919), 19 N. Union St., Middletown, Pa., Op.
GIBSON, R. D.(1907).....310 Wick Ave., Youngstown, O., OALR

GIFFORD, S. R.(1919) . . . .58 E. Washington St., Chicago, Ill., Op.
GILBERT, FRANK Y. . . . . .148 Park St., Portland, Me., OALR
GILL, ARTHUR ERNEST(1928)
U. S. Veterans' Hosp., No. 88, Memphis, Tenn., OALR
*GILL, E. G.(1919) . . . . . .711 S. Jefferson St., Roanoke, Va., ALR
*GILL, G. BACHE(1923)
1624 Eye St., N. W., Washington, D. C., ALR
GILL, GEORGE(1917)
Elyria Savings & Trust Bldg., Elyria, O., OALR
GILL, WM. DAVIS(1929)
323 Medical Arts Bldg., San Antonio, Texas, OALR
GILLETT, WILBUR G. . . . . .201½ N. Main St., Wichita, Kas., Op.
GILLETTE, D. F.(1925) . .State Tower Bldg., Syracuse, N. Y., Op.
*GILLUM, J. R.(1920) . . . . . .221 S. 6th St., Terre Haute, Ind., OALR
GIPNER, J. F.(1926) . . . .53 S. Fitzhugh St., Rochester, N. Y., Op.
GITTENS, T. R.(1920) . . . .401 Davidson Bldg., Sioux City, I., OALR
GLASFORD, S. T.(1924) . . . . . .Box 167, Pekin, Ill., OALR
GLEASON, E. B. . . . . .2112 Pine St., Philadelphia, Pa., ALR
*GLICK, HARRY NAYLOR(1928)
3115 S. Grand Ave., St. Louis, Mo., ALR
*GLOSSER, HERBERT H.(1912)
448 Franklin St., Buffalo, N. Y., Op.
*GLOVER, S. P. . . . . .1118 Twelfth Ave., Altoona, Pa., OALR
GLOWACKI, BEN FRANCIS(1928)
770 Maccabee Bldg., Detroit, Mich., OALR
*GOAR, EVERETT LOGAN(1926)
1300 Walker Ave., Houston, Tex., Op.
GODDARD, H. M. . . . . .1531 Spruce St., Philadelphia, Pa., ALR
GOLDBACK, L. J.(1911), 1012 N. Charles St., Baltimore, Md., Op.
*GOLDING, JOSEPH E.(1925), 170 Hancock St., Brooklyn, N. Y., Op.
GOLDMAN, A. MILTON(1928)
121 E. 60th St., New York, N. Y., Op.
GOLDMAN, H. G. . . . . .57 W. 58 St., New York City, N. Y., ALR
*GOLDSMITH, PERRY G. .510 Medical Arts Bldg., Toronto, Can., ALR
GOLDSTEIN, ISADORE(1929) .1060 Park Ave., New York, N. Y., Op.
*GOLDSTEIN, M. A.(1896), 3858 Westminster Pl., St. Louis, Mo., ALR
GOOD, WM. M.(1929) . . . . . .63 Center St., Waterbury, Conn., ALR
GOODE, ED. A.(1929) . . . .239 Sanford Ave., Flushing, N. Y., ALR
GOODYEAR, H. M.(1924) . . . . . .Doctors Bldg., Cincinnati, O., ALR
GORDON, CHAS. H.(1926) . . . . . .46 Deering St., Portland, Me., ALR
GORDON, GLENN G.(1929) . . . . . .Baker, Ore., ALR
GORDON, J. S.(1919) . .309 Goldsmith Bldg., Milwaukee, Wis., OALR
GORRELL, T. J. H.,
16th and Vincennes Ave., Chicago Heights, Ill., OALR
GOSNEY, C. W.(1920) . .Medical Arts Bldg., Los Angeles, Cal., Op.
*GOTTLIEB, MARK I. . . . . .2 West 87th St., New York, N. Y., ALR
*GOURLEY, G. F.(1916) . . . . . .Sinclair Bldg., Steubenville, O., OALR
*GOUX, RAYMOND SALOT,
545 David Whitney Bldg., Detroit, Mich., OALR

*Gowen, Francis V.(1929)..2033 Pine St., Philadelphia, Pa., ALR
Gowen, Geo. R.(1929)
306 Baker Bldg., Walla Walla, Wash., ALR
*Gracey, Geo. F.....209 State St., Harrisburg, Pa., OALR
*Gradle, H. S.(1911)....58 E. Washington St., Chicago, Ill., Op.
Graham, H. B.....490 Post St., San Francisco, Cal., ALR
Granger, W. B.(1920)
Citizens Natl. Bank Bldg., Emporia, Kan., OALR
Grant, Fred. E.(1912)
Kal. Natl. Bank Bldg., Kalamazoo, Mich., OALR
*Grant, Lee E.(1919)..David Whitney Bldg., Detroit, Mich., OALR
*Grant, P. T.(1919)
1407 Grand Rapids Natl. Bk. Bldg., Grand Rapids, Mich., OALR
Gray, E. B.(1927)
909 Montgomery Bldg., Spartansburg, S. C., ALR
Greear, J. N. Jr.(1929)
1740 M St., N. W., Washington, D. C., Op.
*Green, A. S.
The Green's Eye Hosp., 1801 Bush St., San Francisco, Cal., Op.
Green, John(1905), 3720 Washington Blvd., St. Louis, Mo., Op.
Green, L. D.
The Green's Eye Hosp., 1801 Bush St., San Francisco, Cal., Op.
*Greene, Joseph Berry(1923)
305 Haywood Bldg., Asheville, N. C., ALR
Greene, Laurence Whitridge(1928)
806 Metropolitan Bldg., Denver, Colo., ALR
Greenfield, Samuel D.(1927)
169 New York Ave., Brooklyn, N. Y., ALR
Greenfield, William John(1928)
418 Main St., Hackensack, N. J., ALR
*Greenwood, Allen(1911)
82 Commonwealth Ave., Boston, Mass., Op.
Greever, B. L.(1920)
610 Rorabaugh-Wiley Bldg., Hutchinson, Kan., OALR
Gregg, John B.
612 Security Nat'l Bank Bldg., Sioux Falls, S. D., OALR
Gresser, Ed. B.(1929).....39 Fifth Ave., New York, N. Y., Op.
Griffey, Ed. W.(1929)..1625 Main Blvd., Houston, Texas, ALR
Grim, U. J.....30 N. Michigan Blvd., Chicago, Ill., OALR
*Griscom, J. Milton....2213 Walnut St., Philadelphia, Pa., Op.
Gross, Julius E.(1920)
8030 Jenkins Arcade Bldg., Pittsburg, Pa., ALR
Grosvenor, L. N.(1907), 366 Dakota Ave., So. Huron, S. D., OALR
Grove, W. E.(1914), 120 E. Wisconsin Ave., Milwaukee, Wis., OALR
Grushlow, Israel,
271 Central Pk. W., New York City, N. Y., ALR
Gsell, J. F.(1920).....911 Beacon Bldg., Wichita, Kan., OALR
Guerinot, A. J.(1918)
604 Westinghouse Bldg., Pittsburgh, Pa., OALR

GUGGENHEIM, L. K.....Carleton Bldg., St. Louis, Mo., ALR
GUNDELACH, C. A., 825 University Club Bldg., St. Louis, Mo., ALR
GUNN, M. L.(1929).....Smith Bldg., Harlan, Ky., ALR
GUTHRIE, A. L.(1910)
1010 Medical Arts Bldg., Oklahoma City, Okla., OALR
GUTTMAN, J.....987 Madison Ave., New York City, N. Y., OALR
GUTTMAN, M. R.(1929)...185 N. Wabash Ave., Chicago, Ill., ALR
*GUYTON, B. S.(1918).....Oxford, Miss., OALR
GWINN, G. E.(1916)
704 Central Trust Bldg., San Antonio, Tex., OALR

H

HAAS, FREDERICK J.(1920)
Masonic Bldg., Leavenworth, Kans., OALR
HACKNEY, F. J.(1913)
630 Volunteer Bldg., Chattanooga, Tenn., OALR
HADEN, HENRY C.....1914 Travis St., Houston, Tex., Op.
*HAESSLER, F. HERBERT(1929)
120 E. Wisconsin Ave., Milwaukee, Wis., Op.
HAGEMANN, JOHN A.(1925), Highland Bldg., Pittsburgh, Pa., ALR
HAGLER, E. E.(1896)...401 E. Capitol Ave., Springfield, Ill., OALR
HALE, ARTHUR STANLEY(1928)
1609 Eaton Tower, Detroit, Mich., OALR
*HALL, ALICE K.(1924)..25 E. Washington St., Chicago, Ill., ALR
*HALL, EDWIN PERRY(1928)
State Tower Bldg., Syracuse, N. Y., ALR
HALL, G. C.(1907).....705 Brown Bldg., Louisville, Ky., OALR
HALLETT, G. DEWAYNE(1927)
274 W. 86th St., New York City, N. Y., Op.
HALSTED, FRED S.....738 Metropolitan Bldg., Denver, Colo., OALR
*HAMLIN, FRED EUGENE(1926)
Shenandoah Life Bldg., Roanoke, Va., ALR
HAMPTON, ROBT. R., 804 Boston Bldg., Salt Lake City, Utah, ALR
HANCOCK, J. C.(1914), 135 Cambridge Pl., Brooklyn, N. Y., Op.
Also 2 Rector St., New York, N. Y.,
*HANDS, SIDNEY GILLOTT(1926)
602 1st Nat'l Bank Bldg., Davenport, Ia., ALR
HANEY, WM. P.(1920)
1500 Medical Arts Bldg., Omaha, Neb., OALR
HANS, C. L.(1921).....19 W. 7th St., Cincinnati, O., OALR
HANSEL, FRENCH KELLER(1926)
Beaumont Medical Bldg., St. Louis, Mo., ALR
HANSEN, ERLING W.(1920)
910 Donaldson Bldg., Minneapolis, Minn., OALR
*HANSON, WM. L.(1929).318 Murphy Bldg., E. St. Louis, Ill., ALR
HARALSON, CHAS. H.(1929)
816 Medical Arts Bldg., Tulsa, Okla., Op.
HARBERT, JOHN P.(1918), 135 N. Main St., Bellefontaine, O., OALR

HARDESTY, J. F.(1920).....Missouri Bldg., St. Louis, Mo., Op.
*HARDY, LE GRAND HAVEN(1928)
30 E. 40th St., New York City, N. Y., Op.
HARDY, W. F.(1914).....Humboldt Bldg., St. Louis, Mo., Op.
*HARGITT, CHARLES A.(1926)
17 Schermerhorn St., Brooklyn, N. Y., Op.
*HARKNESS, GORDON F.(1911), Putnam Bldg., Davenport, Ia., OALR
*HARRELL, VOSS(1926).....Hofman Bldg., Detroit, Mich., ALR
*HARRIS, C. M.(1910)
604 Johnstown Trust Bldg., Johnstown, Pa., OALR
*HARRIS, H. B.....Fidelity Med. Bldg., Dayton, O., OALR
HARRIS, THOMAS J.(1929).104 E. 40th St., New York, N. Y., ALR
HARRIS, W. C.(1900).....Groton Bldg., Cincinnati, O., ALR
HARRIS, WM. ROBIN(1923)
500 Lamar Life Bldg., Jackson, Miss., ALR
*HARRISON, FRED HAZARD(1928)
308 First Natl. Bank Bldg., Connellsville, Pa., OALR
HARRISON, W. G.....Empire Bldg., Birmington, Ala., OALR
HARTER, JOHN H.(1929)..320 Stimson Bldg., Seattle, Wash., ALR
HARTLEY, F. A.....302 E. High St., Springfield, O., OALR
HARTSHORNE, ISAAC(1926)
30 W. 59th St., New York City, N. Y., Op.
*HARTZELL, S. M.
Dollar Savings and Trust Co., Youngstown, O., OALR
*HARVEY, N. D.....112 Waterman St., Providence, R. I., OALR
HASELTINE, BURTON(1927)
122 S. Michigan Ave., Chicago, Ill., OALR
HASKELL, ALFRED W.....129 Park St., Portland, Me., Op.
HASKIN, W. H.(1913).....57 W. 57th St., New York, N. Y., ALR
HASPEL, M. DAVID(1911)
603 Maison Blanche Bldg., New Orleans, La., OALR
HASTY, F. E.(1925)....Medical Arts Bldg., Nashville, Tenn., ALR
*HATCH, C. B.(1908).....3 W. Church St., Newark, O., OALR
*HAUER, A. M.(1916).....327 E. State St., Columbus, O., ALR
*HAUGHEY, WILFRED H.(1911)
303 Post Bldg., Battle Creek, Mich., OALR
HAWLEY, C. W.....Medical Arts Bldg., Chicago, Ill., OALR
HAWMAN, ERLE G.....4051 N. Broad St., Philadelphia, Pa., Op.
*HAYDEN, AUSTIN A.(1910)
25 E. Washington St., Chicago, Ill., OALR
*HAYS, HAROLD M.(1912)
64 E. 58th St., New York City, N. Y., ALR
HAZLETT, L. R.(1913)....Savings & Trust Bldg., Butler, Pa., OALR
HEALEY, JOHN F.(1924)..503 Delaware Ave., Buffalo, N. Y., ALR
*HEARD, C. F.(1913).....813 Sassafrass St., Erie, Pa., OALR
HEARD, MARY K...128 19th Ave., North, St. Petersburg, Fla., Op.
HEATH, PARKER(1924), David Whitney Bldg., Detroit, Mich., Op.
HEBERT, ARTHUR W.....450 Sutter St., San Francisco, Cal.,

HECKEL, EDWARD B.(1902)
Jenkins Arcade Bldg,, Pittsburgh, Pa., Op.
HEDRICK, HAROLD B.(1929)
910 Rialto Bldg., Kansas City, Mo., ALR
HEEB, HARRY J.....204 Wisconsin Ave., Milwaukee, Wis., OALR
HEED, C. R.....1205 Spruce St., Philadelphia, Pa., Op.
HEGGIE, N. M.(1905).....Buckman Bldg., Jacksonville, Fla., OALR
HEITGER, J. D.(1905).....808 Francis Bldg., Louisville, Ky., OALR
HEMPSTEAD, BERT E.....Mayo Clinic, Rochester, Minn., ALR
HENNESSEY, WM. W.....333 Essex St., Salem, Mass., OALR
HENNING, CARL....1610 Eye St., N. W., Washington, D. C., Op.
HENNINGER, L. L.(1913)..Professional Bldg., Pasadena, Cal., OALR
HENTON, G. E.(1918).....Morgan Bldg., Portland, Ore., OALR
HERRIMAN, L. L.(1913).....Alamosa, Colo., OALR
HESS, WM. L.(1904).....2423 3rd St., San Diego, Cal., OALR
HETRICK, L. E.(1923), 40 E. 61st St., New York City, N. Y., ALR
HETZEL, C. C.(1906).....Eccles Bldg., Ogden, Utah, OALR
*HICKS, VONNIE MONROE(1926)
Professional Bldg., Raleigh, N. C., Op.
HIGBEE, D. R.(1927).....3245 Fourth St., San Diego, Cal., ALR
HIGGINS, R. P.(1924).....20 Court St., Cortland, N. Y., OALR
*HIGGINS, S. G. ....120 E. Wisconsin Ave., Milwaukee, Wis., OALR
HILL, C. E.(1919).....160 Bloor St., W., Toronto, Can., Op.
*HILL, EMORY(1916).....Professional Bldg., Richmond, Va., Op.
*HILL, FREDERICK THAYER(1928)
Professional Bldg., Waterville, Me., ALR
*HILL, HOWARD FOSTER(1928)
Professional Bldg., Waterville, Me., Op.
HINMAN, E. E.(1924)..146 Washington Ave., Albany, N. Y., ALR
HITZ, H. B.....141 E. Wisconsin Ave., Milwaukee, Wis., ALR
*HOBART, CARL(1929)....Metropolitan Bldg., St. Louis, Mo., Op.
HODSON, B. F.....418 Security Bldg., Miami, Fla., OALR
*HOFFMANN, JOHN N.(1927)....309 Brant Bldg., Canton, O., ALR
HOFFMAN, L. G.(1927)...30 N. Michigan Ave., Chicago, Ill., OALR
HOFFMAN, WALTER F.(1920)
817 Summit Ave., Seattle, Wash., Op.
HOFLING, CHAS. A.(1929).....707 Race St., Cincinnati, O., Op.
*HOFMANN, W. P.(1925)...707 Kahl Bldg., Davenport, Ia., OALR
HOGAN, W. L.(1924).....750 Main St., Hartford, Conn., OALR
*HOGSHEAD, I. M.....602 Georgia Ave., Chattanooga, Tenn., OALR
*HOGUE, D. W.(1908).....34 W. High St., Springfield, O., OALR
HOHF, JULIUS(1915).....Clinic Bldg., Yankton, S. D., OALR
HOLDSWORTH, FRANK
People's Savings Bank Bldg., Traverse City, Mich., OALR
*HOLLOWAY, THOS. B.....1819 Chestnut St., Philadelphia, Pa., Op.
HOLMES, BENJAMIN H.....435 Baker Blk., Racine, Wis., OALR
HOLT, E. E., JR.....723 Congress St., Portland, Me., Op.
HOMPES, I. J.....702 Sharp Bldg., Lincoln, Neb., OALR
*HOOPLE, GORDON D.(1929).Med. Arts Bldg., Syracuse, N. Y., ALR

HOPKINS, W. E.....321 S. Lorraine Blvd., Los Angeles, Cal., OALR
HORN, W. L.(1929)....1040 Park Ave., New York, N. Y., ALR
*HOURN, GEORGE E.(1920)
511 University Club Bldg., St. Louis, Mo., ALR
HOVDE, A. G.(1916)...Board of Trade Bldg., Superior, Wis., OALR
HOWARD, C. N.(1919)....210 S. Indiana St., Warsaw, Ind., OALR
HOWARD, E. F.(1919)
First Natl. Bank Bldg., Vicksburg, Miss., ALR
HOWARD, EDWARD L.(1926)
405 Medical Arts Bldg., Fort Worth, Tex., OALR
*HOWARD, HARVEY J.(1928)
School of Med., Washington University, St. Louis, Mo., Op.
HOWARD, J. W.....Rialto Bldg., Kansas City, Mo., OALR
HOWARD, LLOYD G.(1923)
Council Bluffs Clinic, Council Bluffs, Iowa, OALR
HOWARD, W. H.(1923)
Medical Arts Bldg., Minneapolis, Minn., OALR
HOWE, A. W.(1922)
1616 Washington Bldg., Tacoma, Wash., OALR
HOWE, SPENCER S.(1929)
419 Bellingham Nat'l. Bank Bldg., Bellingham, Wash., ALR
*HUGHES, J. A.(1927).....Sterick Bldg., Memphis, Tenn., OALR
HUGHES, L. W.(1925).....1019 Broad St., Newark, N. J., OALR
HUGHES, RAY WHITBECK(1928)
1501 David Whitney Bldg., Detroit, Mich., OALR
*HUGHES, T. E.(1922)....1000 W. Grace St., Richmond, Va., ALR
*HUGHES, W. F., 401 Hume-Mansur Bldg., Indianapolis, Ind., Op.
*HUGHES, WENDELL L.(1929)
20 E. 53rd St., New York, N. Y., Op.
HUNT, WESTLEY M.(1929), 33 E. 68th St., New York, N. Y., ALR
*HURD, L. M.(1910)....39 E. 50th St., New York City, N. Y., ALR
*HURFF, J. W.(1919).....86 Washington St., Newark, N. J., OALR
HURLEY, E. D.(1914).....419 Boylston St., Boston, Mass., Op.
HURST, V. REEVES(1923), 107 E. Methvin St., Longview, Tex., OALR
*HUSIK, D. N.(1921)....1930 Chestnut St., Philadelphia, Pa., ALR
HUSSEY, E. J.....245 Maple St., Holyoke, Mass., OALR
HUTCHINSON, ABBOTT TRASK(1928)
30 W. 59th St., New York City, N. Y., ALR

I

*IGLAUER, S.....707 Race St., Cincinnati, O., ALR
IMPERATORI, C. J.....17 E. 38th St., New York City, N. Y., ALR
INGERMAN, S. M.(1909)
418 Central Park, W., New York City, N. Y., OALR
INGERSOLL, J. M.(1908), 1700 S. Bayshore Lane, Miami, Fla., OALR
*INGLIS, HARRY J.....43 Bay State Rd., Boston, Mass., ALR
INGRAM, L. C.(1915).....Orlando Clinic, Orlando, Fla., OALR
IRVIN, EDGAR H.....903 First Natl. Bk. Bldg., El Paso, Tex., OALR

IRVINE, A. R.(1918), 1144 Roosevelt Bldg., Los Angeles, Cal., Op.
IRVINE, ROBT. S.(1929)....490 Post St., San Francisco, Cal., ALR
IRWIN, FRANK N.(1905)
114 E. 54th St., New York City, N. Y., Op.
IRWIN, V. J., SR.....1634 Main St., Springfield, Mass., OALR
IRWIN, VINCENT J., JR.(1921)
1634 Main St., Springfield, Mass., OALR
ISRAEL, NORMA E.(1919)
2001 Niels Esperon Bldg., Houston, Tex., OALR
*ISRAEL, SIDNEY....2001 Niels Esperon Bldg., Houston, Tex., ALR
IVINS, H. M.(1910)
815 Merchants Natl. Bank Bldg., Cedar Rapids, Ia., OALR

J

JACKLE, ARTHUR F.(1917), 63 E. Fourth St., Dunkirk, N. Y., Op.
JACKSON, ATRAS EARL(1928)
Med. Arts Bldg., Fort Worth, Tex., Op.
*JACKSON, CHEVALIER.....235 S. 15th St., Philadelphia, Pa., L
JACKSON, J. D.(1920).....232 West 8th St., Erie, Pa., OALR
JACOBS, MAX W.(1916)
Missouri Theatre Bldg., St. Louis, Mo., Op.
JACOBS, S. M.....1018 E. 163rd St., New York City, N. Y., OALR
JACOBY, D. P. A.(1912).....106 Touro St., Newport, R. I., Op.AL
JAMES, J. H.....Natl. City Bank Bldg., Mankato, Minn., OALR
*JAQUISH, CHARLES JOSEPH(1928)
904 Westinghouse Bldg., Pittsburgh, Pa., ALR
JARECKY, HERMAN, 168 W. 86th St., New York City, N. Y., OALR
*JARVIS, DEFOREST C.(1929)..Quarry Bank Bldg., Barre, Vt., ALR
JEAN, GEO. W.....7 East Valerio, Santa Barbara, Cal., Op.
JENKINS, M. C.....223 S. Main St., Pratt, Kansas, ALR
JOBSON, G. B.(1903)
West Park and Liberty Sts., Franklin, Pa., OALR
JOHNSON, A. E.(1919).....Watertown, S. D., OALR
JOHNSON, H. R.(1913)..American Bldg., Fairmont, W. Va., OALR
JOHNSON, OTIS H.....Morehead City, N. C., OALR
JOHNSON, THOMAS HOLLAND(1923)
30 W. 59th St., New York City, N. Y., Op.
JOHNSTON, W. H.(1912)..508 Laurel Bldg., Muscatine, Ia., OALR
*JOHNSTON, WILSON(1917)..611 Lovejoy St., Portland, Ore., OALR
JONES, C. C.(1924).....656 Doctors' Bldg., Cincinnati, O., ALR
*JONES, CECIL CAIN(1928)
1120 Equitable Bldg., Des Moines, Ia., OALR
JONES, EDLEY HICKS(1928)
First Natl. Bank Bldg., Vicksburg, Miss., OALR
*JONES, EDMUND LLOYD(1926)
Wheeling Clinic, Wheeling, W. Va., OALR
JONES, E. L.(1913).First Natl. Bk. Bldg., Cumberland, Md., OALR
JONES, E. W.....44 Atlanta St., Lynn, Mass., Op.A
JONES, F. W.(1919).....Times Bldg., Alton, Ill., OALR
*JONES, L. W.(1925)..16 N. Goodman St., Rochester, N. Y., OALR

*Jones, U. G.(1927)
Jones Eye, Ear Nose & Throat Hos., Johnson City, Tenn., ALR
Jones, Vincent L.(1928)
3511 Washington Ave., St. Louis, Mo., Op.
*Jones, Wm. S.(1927), 522 Sheridan Rd., Menominee, Mich., OALR
Jordan, George T.(1921), 30 N. Michigan Blvd., Chicago, Ill., OALR
*Josephson, Emanuel M.(1929)
993 Park Ave., New York, N. Y., ALR
*Joy, Harold Henry(1926)
504 State Tower Bldg., Syracuse, N. Y., Op.
Joynt, A. J.(1924).....Black Bldg., Waterloo, Ia., OALR
Judd, H. B.(1917)....40 E. 61st St., New York City, N. Y., ALR
Judge, Harry V.(1920).....46 Willett St., Albany, N. Y., Op.

K

Kahn, M. E.(1925)....104 S. Michigan Ave., Chicago, Ill., ALR
Karkeet, Robert Budd(1928)
1203 Selling Bldg., Portland, Ore., OALR
Kauffman, A. B.(1922)
1000 Chimes Bldg., Syracuse, N. Y., ALR
*Kaufman, A. Spenser(1917)
1923 Spruce St., Philadelphia, Pa., ALR
Kearby, D. O.(1917)
709 Hume-Mansur Bldg., Indianapolis, Ind., ALR
Keating, T. F.(1927)
1042 Maccabees Bldg., Detroit, Mich., OALR
Keefe, Wm. J.(1929), 110 E. Sidney Ave., Mt. Vernon, N. Y., ALR
Keeler, J. C.....Med. Arts Bldg., Philadelphia, Pa., ALR
*Keim, W. F.(1924)....25 Roseville Ave., New York, N. J., ALR
Keith, W. E.....Waldheim Bldg., Kansas City, Mo., OALR
Keller, S. A.....Sioux Falls Clinic, Sioux Falls, S. D., OALR
Kelley, Isaac D., Jr.(1928)
811 Beaumont Med. Bldg., St. Louis, Mo., ALR
Kelly, Joseph Dominic(1928)
114 E. 54th St., New York City, N. Y., OALR
*Kemler, Jos.(1922).....1908 Eutaw Pl., Baltimore, Md., OALR
Kendall, M. R.(1919).....Osborn Bldg., Cleveland, O., ALR
Kennedy, Eldred Weston(1928)
707 Gas & Elect. Bldg., Rochester, N. Y., Op.
Kenney, Wm. L.....Corby Forsee Bldg., St. Joseph, Mo., OALR
Kennon, W. G.(1924)....Doctors' Bldg., Nashville, Tenn., OALR
Kettelkamp, Fred O.(1926)
301 Ferguson Bldg., Colorado Springs, Colo., ALR
Key, Ben Witt(1914)
100 W. 59th St., New York City, N. Y., Op.
Key, Sam N.....Norwood Bldg., Austin, Tex., OALR
Keys, Morton J.(1921)
100 Jones Blk., Victoria, B. C., Canada, OALR

KIEFER, HUGO A.(1915), Brockman Bldg., Los Angeles, Cal., OALR
KIEHLE, F. A.....410 Med. Arts Bldg., Portland, Ore., OALR
KILBOURNE, P. H.(1924)..Fidelity Med. Bldg., Dayton, O., OALR
KILLEEN, JOHN JOSEPH(1928)
104 S. Michigan Ave., Chicago, Ill., ALR
*KINCAID, J. H.....421 W. Church Ave., Knoxville, Tenn., OALR
KINCANNON, LEROY TAYLOR(1928)
The Woodward, Birmingham, Ala., OALR
*KING, CLARENCE(1910), Union Central Bldg., Cincinnati, O., Op.
KING, CHAS. R.(1920).....Spitzer Bldg., Toledo, O., OALR
*KING, ED.....707 Race St., Cincinnati, O., ALR
*KING, G. L.....537 E. Market St., Alliance, O., OALR
KING, GEO. L., JR.(1929).....537 Market St., Alliance, O., Op.
KING, J. J.(1916)....30 E. 40th St., New York City, N. Y., ALR
*KING, O. H.(1916)..Dugan-Stuart Bldg., Hot Springs, Ark., OALR
KINNE, SANFORD HALSOY(1928)
73 Main St., Binghampton, N. Y., ALR
*KIRBY, D. B.(1924)..30 W. 59th St., New York City, N. Y., Op.
KIRKPATRICK, SAMUEL.....902 Alabama Ave., Selma, Ala., OALR
KISER, J. D.....Central Natl. Bank Bldg., Bartlesville, Okla., OALR
*KISTLER, O. F.....43 N. Franklin St., Wilkes-Barre, Pa., Op.AR
KISTNER, F. B.....600 Mayer Bldg., Portland, Ore., OALR
KITTELSON, THEODORE(1915).....Fergus Falls, Minn., OALR
*KLEINERT, MARGARET NOYES
82 Commonwealth Ave., Boston, Mass., ALR
KLINE, H. G.(1912)....State Tower Bldg., Syracuse, N. Y., ALR
*KLINEDINST, JOHN FERD.(1906)
220 S. George St., York, Pa., Op.A
KLINGNER, THOS. O.(1920)
318 Landers Bldg., Springfield, Mo., OALR
KNAPP, ARNOLD.....10 E. 54th St., New York City, N. Y., Op.
KNAPP, B.....Cleveland Life Bldg., Evansville, Ind., OALR
*KNAPP, F. N.(1925).....Bradley Bldg., Duluth, Minn., OALR
*KNEEDLER, G. C.(1912)..Professional Bldg., Pittsburgh, Pa., ALR
KNODE, A. R.(1916)....708 Med. Arts Bldg., Omaha, Nebr., ALR
KNOWLTON, CHAS. D.(1929)
520 Commonwealth Ave., Boston, Mass., ALR
*KOEBBE, EZRA EDWIN(1929).....Columbus, Nebr., ALR
*KOENIG, IVAN J.(1928).....40 North St., Buffalo, N. Y., Op.
KOLLOCK, C. W.....86 Wentworth St., Charleston, S. C., OALR
KOOBS, H. J. G.(1920)..117½ W. Walnut St., Rogers, Ark., OALR
*KOPETZKY, S. J.....51 W. 73rd St., New York City, N. Y., ALR
KRAFT, OSCAR H.....1518 N. Dearborn Pkwy., Chicago, Ill., Op.
*KRAUSS, FREDERICK....1703 Chestnut St., Philadelphia, Pa., OALR
*KREBS, ADOLPH(1914).....Jenkins Arcade, Pittsburgh, Pa., Op.
KRESS, GEO. H.....245 Bradbury Bldg., Los Angeles, Cal., OALR
KRIEGER, G. M.....701 Washington St., Michigan City, Ind., OALR
*KRIEGER, WM. A.(1919), 35 Market St., Poughkeepsie, N. Y., OALR

KRIMSKY, JOSEPH (1928)
263 Eastern Parkway, Brooklyn, N. Y., OALR
KRUG, ERNEST F. (1908)
12 W. 44th St., New York City, N. Y., Op.
KRUSKAL, I. D. (1925) ....863 Prospect Pl., Brooklyn, N. Y., Op.
*KUHN, HUGH A. (1928) ....1st Trust Bldg., Hammond, Ind., ALR
KULLY, B. M. (1922) .....Med. Arts Bldg., Omaha, Neb., ALR

L

*LAESSLE, HENRY A. (1922)
The Lenox, 13th & Spruce Sts., Philadelphia, Pa., ALR
LA FORCE, B. D. .....1140 Beverley Way, Altadena, Cal., OALR
LA FORCE, E. F. (1908)
Iowa State Bank Bldg., Burlington, Ia., OALR
LAMB, HARVEY D. (1920)
826 Metropolitan Bldg., St. Louis, Mo., Op.
LAMB, HARRY H. (1929)
American Bank Bldg., Davenport, Ia., ALR
LAMB, ROBT. SCOTT .....Stoneleigh Ct., Washington, D. C., Op.
*LANCASTER, W. B. (1913)
520 Commonwealth Ave., Boston, Mass., Op.
LANDES, GEORGE A. .....Parsons, Kas., OALR
LANDFRIED, C. J. .....607 Baronne Bldg., New Orleans, La., ALR
LANING, J. H. (1918) ....907 Rialto Bldg., Kansas City, Mo., ALR
LAPSLEY, ROBERT M. (1902) ..Masonic Temple, Keokuk, Ia., OALR
*LARGE, S. H. (1904) .....Hanna Bldg., Cleveland, O., ALR
LARKIN, BERNARD J. (1916)
Hume-Mansur Bldg., Indianapolis, Ind., Op.
LARSEN, CARL L. (1916)
1027 Lowry Medical Bldg., St. Paul, Minn., OALR
*LARUE, C. L. (1922) .....Highland Clinic, Shreveport, La., OALR
LASSALLE, ALBERT (1924), 3439 St. Denis St., Montreal, Can., OALR
LASSEN, FRITZ (1915) .....Pueblo Clinic, Pueblo, Colo., OALR
LA VIGNE, A. A. .....471 Park Ave., New York City, N. Y., OALR
LAWRENCE, GRANVILLE A. ..
Medical Arts Bldg., Philadelphia, Pa., Op.
LAWWILL, STEWART (1928)
First Natl. Bldg., Chattanooga, Tenn., OALR
LEA, JOS. A. .....1125 Rialto Bldg., Kansas City, Mo., ALR
LEAVY, C. A. .....Metropolitan Bldg., St. Louis, Mo., ALR
LEBENSOHN, JAMES E. (1927)
25 E. Washington, Chicago, Ill., OALR
LE COMPTE, E. D. (1927)
627 Boston Bldg., Salt Lake City, Utah., OALR
LEDERER, FRANCIS L. (1925)
30 N. Michigan Blvd., Chicago, Ill., ALR
LEECH, J. W. .....369 Broad St., Providence, R. I., OALR
LEFLER, ANNA B. (1918), 610 S. Broadway, Los Angeles, Cal., OALR

LEHAN, JAMES W.....Park Place-8th St., Greeley, Colo., OALR
LEJEUNE, F. E.(1927)
632 Maison Blanche Bldg., New Orleans, La., ALR
LEMERE, H. B.(1912)....Medical Arts Bldg., Omaha, Neb., OALR
*LEMOINE, A. N.(1920)..1118 Rialto Bldg., Kansas City, Mo., Op.
LENKER, JOHN N.(1908)....Osborne Bldg., Cleveland, O., ALR
LENT, E. J.....122 N. Lafayette Blvd., South Bend, Ind., OALR
*LERNER, MACY LEVI(1923), 332 Park Ave., Rochester, N. Y., Op.
*LESTER, HARRY S.(1909)....420 Murray Bldg., Streator, Ill., OALR
LEVENGOOD, H. W.(1927)
327 Wilshire Blvd., Santa Monica, Cal., OALR
*LEVY, LOUIS(1916)..1052 Madison Ave., Memphis, Tenn., ALR
LEVY, ROBERT(1896)....Metropolitan Bldg., Denver, Colo., ALR
*LE WIN, THURBER(1929).112 Linwood Ave., Buffalo, N. Y., Op.
LEWIS, A. C.(1916).....Exchange Bldg., Memphis, Tenn., OALR
*LEWIS, EUGENE R.(1926), Roosevelt Bldg., Los Angeles, Cal., ALR
LEWIS, FIELDING O.....259 S. 17th St., Philadelphia, Pa., ALR
LEWIS, F. PARK.....454 Franklin St., Buffalo, N. Y., Op.
LEWIS, GEORGE A.(1920).....Wall Bldg., Roundup, Mont., OALR
LEWIS, J. D.(1921)....1534 Estado St., San Barbara, Cal., OALR
LEWIS, J. P. (1915).....543 Boylston St., Boston, Mass., ALR
LEWIS, PHILIP MERIWETHER(1928)
Exchange Bldg., Memphis, Tenn., OALR
LEWIS, WILLIAM W.(1921)
836 Lowry Bldg., St. Paul, Minn., OALR
LEYDA, JAMES H.(1927), Metropolitan Bldg., Denver, Colo., ALR
LICHTENBERG, J. S.(1906)..Rialto Bldg., Kansas City, Mo., Op.
LIDIKAY, C. J.(1920)....903 N. 7th St., Kansas City, Kans., OALR
LIERLE, DEAN MCALLISTER(1928)
University Hosp., Iowa City, Ia., ALR
LIFSCHUTZ, JACOB(1925), 310 S. Michigan Ave., Chicago, Ill., OALR
LILLIE, HAROLD I.....Mayo Clinic, Rochester, Minn., ALR
*LINGEMAN, ED. L.(1929)....406 Pennway Bldg., Indianapolis, Ind.,
*LINN, J. G.(1919), 7026 Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
*LISCHKOFF, M. A.(1913)....Blount Bldg., Pensacola, Fla., OALR
LITCHFIELD, J. T.(1921)
609 Yeates Bldg., Minneapolis, Minn., OALR
LLOYD, A. W.(1915).....804 Lloyd Bldg., Hammond, Ind., OALR
*LLOYD, RALPH I.(1925)....14 Eighth Ave., Brooklyn, N. Y., Op.
LOEB, CLARENCE(1907)
1054 Tower Rd., Hubbard Woods, Ill., Op.
*LOFTUS, JOHN EDWARD(1921)
605 Medical Arts Bldg., Philadelphia, Pa., ALR
LOKEY, H. M.....Medical Arts Bldg., Atlanta, Ga., OALR
*LOMBARDO, MELCHIORE(1923)
142 Joralemon St., Brooklyn, N. Y., Op.
LOOMIS, E. A.(1916)
Medical Arts Bldg., Minneapolis, Minn., OALR

LOOPER, EDWARD ANDERSON (1924)
104 Madison St., Baltimore, Md., OALR
LORIE, ALVIN (1920) . . . . Commerce Bldg., Kansas City, Mo., ALR
LORING, J. B. . . . . 25 E. Washington St., Chicago, Ill., OALR
LOVE, JOHN G. (1920) . . . . . Ilgenfritz Bldg., Sedalia, Mo., OALR
LOWRY, B. W. (1925) . . 408 Citrus Exch. Bldg., Tampa, Fla., OALR
LUBMAN, MAX (1919), 17 E. 96th St., New York City, N. Y., ALR
LUCAS, HAROLD ROY (1928)
Medical Dental Bldg., Portland, Ore., OALR
LUEDDE, W. H. (1916) . . Metropolitan Bldg., St. Louis, Mo., Op.
*LUKENS, CHARLES (1906) . . . . . 316 Michigan St., Toledo, O., OALR
*LUKENS, ROBT. McD. . . . . 1923 Spruce St., Philadelphia, Pa., ALR
LUNDON, A. E. (1927), 1501 Crescent St., Montreal, Canada, ALR
LUPTON, IRVING MARTIN (1928)
Medical Arts Bldg., Portland, Ore., OALR
LUX, PAUL (1925) . . 409 Commerce Bldg., Kansas City, Mo., ALR
LYLE, DONALD J. (1927) . . . . . 19 W. 7th St., Cincinnati, O., OALR
*LYMAN, HARRY W. (1923), 906 Carleton Bldg., St. Louis, Mo., ALR
LYNCH, R. C. (1913)
Maison Blanche Bldg., New Orleans, La., ALR
LYONS, H. R. . . . . 30 N. Michigan Blvd., Chicago, Ill., ALR

M

MCALESTER, ANDREW W., JR.,
625 Bryant Bldg., Kansas City, Mo., Op.
MCALLISTER, J. C. (1910) . . . . 10 S. Mill St., Ridgeway, Pa., AL
MCBEAN, GEO. M. . . . . 58 E. Washington, Chicago, Ill., OALR
*MCBRIDE, W. O. (1917)
2902 S. Fairfield Ave., Ft. Wayne, Ind., OALR
MCCANNEL, A. D. . . . . 33 Central Ave., W., Minot, N. D., OALR
MCCARTNEY, OLIVER P. (1920)
707 Huron Bldg., Kansas City, Kan., OALR
MCCARTY, VIRGIL W. . . . . 800 Rialto Bldg., Kansas City, Mo., OALR
*MCCASKEY, C. H., Continental Bk. Bldg., Indianapolis, Ind., ALR
MCCAW, JOHN A. . . . . 418 Majestic Bldg., Denver, Colo., Op.
MCCLELLAN, W. E. (1927), 101 Haywood St., Asheville, N. C., OALR
MCCLELLAND, CARL C. (1923)
1857 David Whitney Bldg., Detroit, Mich., OALR
MCCONNELL, T. E.
Logan Trust Bldg., New Kensington, Pa., OALR
*MCCOOL, J. L. (1911) . . . . 450 Sutter St., San Francisco, Cal., Op.
MCCOY, J. . . . . 730 Fifth Ave., New York City, N. Y., ALR
MCCREADY, J. H. . . . . 121 University Pl., Pittsburgh, Pa., ALR
MCCULLOCH, R. J. P. (1920)
830 Medical Arts Bldg., Toronto, Can., OALR
*MCCULLOUGH, C. J. (1929)
Washington Trust Bldg., Washington, Pa., ALR

*McDannald, C. E.(1927)
100 W. 59th St., New York City, N. Y., Op.
McDavitt, Bertha Schenck Stecker(1929)
Woodson Eye, Ear, Nose Hosp., Temple, Texas, ALR
*McDermott, John J.(1924)
1821 Niles Ave., St. Joseph, Mich., OALR
McDiarmid, H. Oliver(1918)
35 Clement Block, Brandon, Manitoba, Can., OALR
McDonald, C. L.....964 Rose Bldg., Cleveland, O., OALR
McDonald, Dennis J.(1926)
17 E. 38th St., New York City, N. Y., ALR
*McDowell, N. D.(1914)
275 Alexander St., Rochester, N. Y., OALR
McFarlane, Murray.....190 Bloor St., E., Toronto, Can., OALR
*McGarvey, Wm. Ed.(1929)
Jackson City Bank, Jackson, Mich., ALR
*McGinnis, Edwin.....104 S. Michigan Blvd., Chicago, Ill., ALR
*McGivern, C. S.(1922)
101 S. Indiana Ave., Atlantic City., N. J., ALR
*McGuire, H. H.....105 N. Braddock St., Winchester, Va., OALR
McHenry, D. D.(1912)
604 Medical Arts Bldg., Oklahoma City, Okla., OALR
McKee, Claud W.(1917)..Coulter Bldg., Greensburg, Pa., OALR
*McKee, Geo. J.(1922), Westinghouse Bldg., Pittsburgh, Pa., ALR
*McKee, Joseph Wallace(1928)
1222 Rialto Bldg., Kansas City, Mo., Op.
*McKee, Otto S.....83 Bryant St., Buffalo, N. Y., ALR
*McKee, S. Hanford(1910)
1528 Crescent St., Montreal, Can., Op.
McKelvey, A. D.....160 Bloor St., W., Toronto, Can., ALR
McKennan, Jas. W.
514 Washington Trust Bldg., Washington, Pa., Op.
*McKeown, Elmer F.....Metropolitan Bldg., Denver, Colo., OALR
*McKinney, A. R.(1922)
704 Second Natl. Bk. Bldg., Saginaw, Mich., OALR
McKinney, R.....899 Madison Ave., Memphis, Tenn., ALR
McLaughlin, P. A.,
71 S. Washington St., Wilkes-Barre, Pa., OALR
McLaurin, John G.(1926)
Medical Arts Bldg., Dallas, Texas, OALR
McLoone, John J.(1926)..611 Heard Bldg., Phoenix, Ariz., OALR
McMahon, Bernard John(1926)
Missouri Theatre Bldg., St. Louis, Mo., ALR
*McMurray, J. B., Washington Trust Bldg., Washington, Pa., OALR
*McNally, William James
Medical Arts Bldg., Montreal, Canada, ALR
*McNichols, W. A.(1925)
Dixon Nat'l. Bk. Bldg., Dixon, Ill., OALR
*McReynolds, G. S.....Professional Bldg., Temple, Tex., ALR

McREYNOLDS, JOHN O.,
907 Mercantile Bk. Bldg., Dallas, Tex., OALR
*McWILLIAMS, CHAS. A.(1927)
G. and S. I. Bldg., Gulfport, Miss., ALR
MACFARLANE, P. B., 152 James St., S. Hamilton, Ont., Can., OALR
*MACKENZIE, ALICE V.(1919)
1724 Spruce St., Philadelphia, Pa., OALR
*MACKENZIE, GEO. W.(1912)
1724 Spruce St., Philadelphia, Pa., OALR
MacLACHLAN, A. A.....May Bldg., Pittsburgh, Pa., ALR
MacMILLAN, J. A.(1925), 1410 Stanley St., Montreal, Can., Op.
MACNIE, J. S.....74 S. 11th St., Minneapolis, Minn., OALR
MADDEN, REED.....Allen Bldg., Xenia, O., OALR
MAGGARD, D. I.(1920)..715 Orpheum Bldg., Wichita, Kan., OALR
MAGHY, CHAS. A.(1926)
Wilshire Medical Bldg., Los Angeles, Cal., Op.
MAHONEY, PAUL LEO(1928)
Donaghey Bldg., Little Rock, Ark., ALR
MAIDEN, S. D.(1918), 403 Bennett Bldg., Council Bluffs, Ia., OALR
MAIRE, LEWIS E.(1906)..710 Kresege Bldg., Detroit, Mich., OALR
MALONEY, THOS. J.....1032 Lowry Bldg., St. Paul, Minn., OALR
*MANN, R. H. T.(1913)
State Natl. Bk. Bldg., Texarkana, Tex., OALR
*MARCH, CLARA A.(1922), 465 Ashland Ave., Buffalo, N. Y., Op.
MARKEL, J. CLYDE.....Westinghouse Bldg., Pittsburgh, Pa., Op.
*MARKS, JULIAN B.(1929)
1st Nat'l. Bank Bldg., Milwaukee, Wis., ALR
MARKS, SAMUEL BLACKBURN(1928)
Sec. Trust Bldg., Lexington, Ky., ALR
*MARLOW, SEARLE B.(1924)
1004 State Tower Bldg., Syracuse, N. Y., Op.
MARQUIS, GEO. P.....30 N. Michigan Blvd., Chicago, Ill., ALR
MARSHALL, GEORGE GUERIN....Gryphon Bldg., Rutland, Vt., OALR
MARSHALL, WATSON.....121 University Pl., Pittsburgh, Pa., ALR
MARTIN, ROY D.(1929).....Foote Bldg., Hastings, Nebr., ALR
MASON, ROY EARL(1928).....Frisco Bldg., St. Louis, Mo., Op.
*MASTERS, ROBERT JOHN(1928)
23 E. Ohio St., Indianapolis, Ind., Op.
MATHENY, R. C.
405 Bk. of Galesburg Bldg., Galesburg, Ill., OALR
MATHESON, J. P.(1915)....6 West 7th St., Charlotte, N. C., ALR
MATTHEWS, JUSTUS
1021 Metropolitan Bk. Bldg., Minneapolis, Minn., LR
MAUDLIN, L. O.
Chamber of Commerce Bldg., Greenville, S. C., OALR
MAUMENEE, A. E.....711 Empire Bldg., Birmingham, Ala., OALR
MAXEY, E. E.(1911)....609 Becker Bldg., Aberdeen, Wash., OALR
MAY, CHAS. H.(1904)
698 Madison Ave., New York City, N. Y., Op.

MAY, J. V.(1915).....1703 Main St., Marinette, Wis., OALR
MAYBAUM, J. L.(1916)
1079 Park Ave., New York City, N. Y., ALR
MAYER, LEO LOEB(1928)
104 S. Michigan Ave., Chicago, Ill., Op.
MAYERBERG, EMIL R.(1929)
Medical Arts Bldg., Wilmington, Del., ALR
MEANOR, WM. C...619 Westinghouse Bldg., Pittsburgh, Pa., Op.
MEIERHOF, E. L.(1912), 12 E. 86th St., New York City, N. Y., OALR
MELLINGER, WM. J.(1927)
120 E. Sola St., Santa Barbara, Cal., ALR
MENGER, LOUIS C.(1929).202 Weirfield St., Brooklyn, N. Y., ALR
MERCER, RAY(1920).....W. C. U. Bldg., Quincy, Ill., OALR
MERCER, R. E.....David Whitney Bldg., Detroit, Mich., LR
MERKEL, CHARLES CARSON(1928)
1409 David Whitney Bldg., Detroit, Mich., ALR
MERRILL, H. G.(1917)
35 East 2nd South St., South, Provo, Utah, OALR
MERRILL, W. HOWE(1906)
Bay State Bldg., Lawrence, Mass., OALR
*MERTINS, PAUL STEARNS(1928)
1115 Bell Bldg., Montgomery, Ala., OALR
*MESSIER, A. E.(1925).....390 Main St., Worcester, Mass., OALR
MESSINGER, H. C.....210 Angell St., Providence, R. I., Op.
METZ, ROY B.....1214 Guardian Bldg., Cleveland, O., Op.
*METZENBAUM, MYRON(1912)....Rose Bldg., Cleveland, O., ALR
MEYER, SAMUEL JAMES(1928)
58 E. Washington St., Chicago, Ill., Op.
MIDDLETON, A. B.....Sterry Block, Pontiac, Ill., OALR
MIDDLETON, HARRY E.(1920).....Times Bldg., Alton, Ill., OALR
MILLER, ALICE R.(1919)
505 Guaranty Bldg., West Palm Beach, Fla., OALR
MILLER, C. E.....305 Western Reserve Bldg., Muncie, Ind., OALR
MILLER, CLIFTON M., Stuart Circle Hospital, Richmond, Va., OALR
*MILLER, E. B.....2008 Walnut St., Philadelphia, Pa., Op.
*MILLER, HUGH.....Chambers Bldg., Kansas City, Mo., OALR
MILLER, J. W.(1929)....305 E. 17th St., New York, N. Y., ALR
MILLER, VERNON B.(1928)
810 Metropolitan Bldg., St. Louis, Mo., ALR
MILLETTE, J. W.....Reibold Bldg., Dayton, O., OALR
MINER, S. G.(1896).....2641 E. Jefferson, Detroit, Mich., ALR
*MINOR, C. L.....First Natl. Bank Bldg., Springfield, O., OALR
MINOR, H. F.....Randolph Bldg., Memphis, Tenn., OALR
MINSKY, HENRY(1926)
215 W. 88th St., New York City, N. Y., Op.
MINTON, WM. H....First State Bank Bldg., St. Joseph, Mo., OALR
*MITCHELL, E. K.(1915)
710 W. Lehigh Ave., Philadelphia, Pa., ALR
*MITHOEFER, W.....19 Garfield Pl., Cincinnati, O., ALR

MITTENDORF, A, D.....339 Park Ave., New York City, N. Y., Op.
MITTENDORF, W. KEITH,
115 E. 53rd St., New York City, N. Y., Op.
MOISE, ALLYN B.(1918), Medical Arts Bldg., Shreveport, La., ALR
MOLT, WM. F.(1911)
529 Bankers Trust Bldg., Indianapolis, Ind., ALR
MONAGHAN, D. G.....209 16th St., Denver, Colo., Op.
*MONAHAN, J. A.(1913)....181 Chestnut St., Clinton, Mass., OALR
MONOSMITH, O. B.(1912).....O. P. S. Bldg., Lorain, O., OALR
*MONSON, S. H.....1322 B. F. Keith Bldg., Cleveland, O., OALR
MONTGOMERY, DANIEL CAMERON(1928)
Greenley Bldg., Greenville, Miss., ALR
*MOORE, G. A.(1915).....353 Main St., Palmer, Mass., OALR
MOORE, GEO. H.(1919)
18th and Chestnut Sts., Philadelphia, Pa., OALR
*MOORE, JOHN D.(1925), 6 Washington St., Bloomfield, N. J., OALR
*MOORE, ROY SEELEY(1928)
713 E. Genesee St., Syracuse, N. Y., ALR
*MOORE, T. K.....416 Central Sav. & Trust Bldg., Akron, O., OALR
MOORE, T. W.(1901)..1050 Fifth Ave., Huntington, W. Va., OALR
MOORE, WILLIAM FREDERICK
255 S. 16th St., Philadelphia, Pa., ALR
MORGAN, JAMES A.(1916)
48 Young Bldg., Honolulu, T. Hawaii, OALR
*MORIARTY, RICHARD W.(1928)
57 Maple Ave., White Plains, N. Y., OALR
MORRIS, A. G.(1918)...303 Alexander St., Rochester, N. Y., ALR
MORRISON, WM. WALLACE(1926)
39 E. 50th St., New York City, N. Y., ALR
MORSE, A. W.....507 Phoenix Bldg., Butte, Mont., OALR
MORSMAN, L. W.(1920)
Care of Morsman Clinic, Hibbing, Minn., Op.
*MORTON, HOWARD McI.
1111 Nicollet Ave., Minneapolis, Minn., Op.
MORTON, J. B.(1924)....90 E. Warren Ave., Detroit, Mich., OALR
*MOSHER, HARRIS P.....828 Beacon St., Boston, Mass., ALR
MOSS, HARVEY E.....1321 Rialto Bldg., Kansas City, Mo., OALR
MOTT, JOHN S.....214 Argyle Bldg., Kansas City, Mo., OALR
MOTTO, M. P.(1929).....Rose Bldg., Cleveland, Ohio, Op.
MOULTON, EVERETT C.
Merchants Natl. Bank Bldg., Ft. Smith, Ark., OALR
MOULTON, H.(1900)
Merchants Natl. Bank Bldg., Ft. Smith, Ark., OALR
*MULLIN, WM. V.(1913)
Cleveland Clinic, Euclid Ave. at 93rd St., Cleveland, O., ALR
MULSOW, J. E.(1921)
221 Wisconsin Ave., W., Milwaukee, Wis., OALR
*MUNCASTER, S. B...1026 16th St., N. W., Washington, D. C., Op.
*MUNCY, WM. M.(1914)..162 Angell St., Providence, R. I., OALR

*MUNDT, G. H.(1912)....25 E. Washington St., Chicago, Ill., OALR
MURDOCK, J. F.....Bessemer Bldg., Pittsburgh, Pa., Op.
MURPHY, W. E.....707 Race St., Cincinnati, O., OALR
MURRAY, A. N.....4753 Broadway, Chicago, Ill., OALR
MUSSUN, W. G.(1924).....Hanna Bldg., Cleveland, O., ALR
MYERS, ELMER A., 407 Board of Trade Bldg., Superior, Wis., OALR
MYERS, EUGENE LEE(1926), 207 Wall Bldg., St. Louis, Mo., ALR
*MYERS, JOHN L.(1920), 815 Shukert Bldg., Kansas City, Mo., OALR
MYERSON, M. C.(1923)....198 Lincoln Pl., Brooklyn, N. Y., ALR

N

NADEAU, E. G.....Bellin Bldg., Green Bay, Wis., OALR
NAFTZGER, J. B.....Davidson Bldg., Sioux City, Ia., OALR
NANCE, WILLIS O.....30 N. Michigan Blvd., Chicago, Ill., OALR
NASH, C. STEWART(1929)
26 S. Goodman St., Rochester, N. Y., ALR
NEEPER, EDW. R.,
Ex. Nat'l. Bank Bldg., Colorado Springs, Colo., Op.
NEHER, EDWIN MANSON(1926)
Boston Bldg., Salt Lake City, Utah, Op.
NEIGHBORS, CLARENCE A.
120 S. Los Angeles S., Anaheim, Cal., OALR
*NELSON, CHAS. F.....606 Schofield Bldg., Cleveland, O., OALR
NELSON, LOUIS A.....830 Lowry Bldg., St. Paul, Minn., Op.
NELSON, LLOYD L.(1923)....Fleming Bldg., Norfolk, Neb., OALR
NEMOURS, PAUL R.(1929)..3852 Greer Ave., St. Louis, Mo., ALR
*NEUMANN, WILLIAM HENRY(1928)
1005 N. 8th St., Sheboygan, Wis., OALR
NEW, GORDON B.....Mayo Clinic, Rochester, Minn., L
NEWCOMB, JOHN R.
410 Hume-Mansur Bldg., Indianapolis, Ind., Op.
NEWELL, WM. S.....1029 Vermont Ave., Washington, D. C., Op.
*NEWHART, HORACE(1905)
Medical Arts Bldg., Minneapolis, Minn., ALR
NEWTON, F. H.(1925)....907 Mercantile Bldg., Dallas, Tex., OALR
NEWTON, L. A.(1919)
609 Medical Arts Bldg., Oklahoma City, Okla., OALR
NISSELSON, MAX.....754 Beck St., New York City, N. Y., ALR
NOONAN, W. J.(1905).....4753 Broadway, Chicago, Ill., OALR
*NORCROSS, E. P.(1924).....8 S. Michigan Ave., Chicago, Ill., ALR
NORTHCOTT, THOMAS A.(1926)
17 E. 38th St., New York City, N. Y., Op.
*NOVAK, FRANK J., JR.....30 N. Michigan Blvd., Chicago, Ill., ALR
NUTTER, C. F.....16 Amherst St., Nashua, N. H., Op.

O

OAST, SAMUEL P.(1929)..30 E. 40th St., New York, N. Y., Op.
OBERG, C. M.....Syndicate Bldg., Minneapolis, Minn., OALR

*O'Brien, C. S.(1924)....University Hospital, Iowa City, Ia., Op.
O'Brien, Steve A.(1916)
1st Nat'l. Bank Bldg., Mason City, Ia., OALR
*O'Connor, D. F.(1918).....671 Broad St., Newark, N. J., OALR
O'Connor, Roderick P.....1904 Franklin St., Oakland, Cal., Op.
Odeneal, Thomas H.(1929)..234 Cabot St., Beverly, Mass., OALR
Oertel, T. E.....S. F. Bldg., Augusta, Ga., OALR
Oesterling, H. E.(1922)
642 N. Interlachen Ave., Winter Park, Fla., OALR
*Offutt, W. N.(1905)
406 Security Trust Bldg., Lexington, Ky., OALR
*Olsho, S. L.(1912).....235 S. 15th St., Philadelphia, Pa., OALR
Orcutt, D. S.(1912)....30 N. Michigan Ave., Chicago, Ill., Op.
*O'Reilly, Charles A.(1911)
521 Med. Arts Bldg., Philadelphia, Pa., ALR
*O'Reilly, W. F.(1913)....44 S. Common St., Lynn, Mass., OALR
*O'Rourke, D. H.(1927)...217 Imperial Bldg., Denver, Colo., Op.
Orr, Adrian V. B.(1929).....525 Welsh St., Chester, Pa., ALR
Orton, Henry B.....24 Commerce St., Newark, N. J., ALR
O'Sullivan, T. J.(1927).....10 Pine St., Portland, Me., ALR
Overman, F. V.....Hume-Mansur Bldg., Indianapolis, Ind., ALR
Owen, F. S.....Medical Arts Bldg., Omaha, Neb., OALR

P

Packard, Louis(1926).....Denver, Colo., OALR
*Painter, A. M.(1918)..418 Bryant Bldg., Kansas City, Mo., ALR
*Palmer, Arthur(1926)
667 Madison Ave., New York City., N. Y., ALR
Palmer, Bascom Headen(1928)
Roney Med. Clinic, 23rd St. at Collins, Miami Beach, Fla., OALR
Palmer, Edw. M.(1919)..629 Beacon Bldg., Wichita, Kan., OALR
Palmer, F. E.(1928).....P. O. Box 267, Sterling, Colo., OALR
Park, J. Walter(1910), 32 N. Second St., Harrisburg, Pa., OALR
Parker, Walter R.
1st Nat'l. Bank Bldg., Ann Arbor, Mich., Op.
Parramore, James B.(1929)
322 St. James Bldg., Jacksonville, Fla., ALR
Parrish, Robert E.
Medical Arts Bldg., San Antonio, Texas, ALR
Parsons, J. G...313 Bank Electric Bldg., Lewistown, Mont., OALR
Patterson, Ellen J.(1917)
121 University Pl., Pittsburgh, Pa., ALR
*Patterson, W. E.....74 S. 11th St., Minneapolis, Minn., OALR
*Patton, J. M.(1910).....Medical Arts Bldg., Omaha, Neb., Op.
Payne, A. L.(1908)
402½ S. Barston St., Eau Claire, Wis., OALR
Payne, R. J.(1925)..University Club Bldg., St. Louis, Mo., ALR
Pearlman, S. J.....180 N. Michigan Ave., Chicago, Ill., ALR

PEARSON, WM. W.(1908)
417 Bankers Trust Bldg., Des Moines, Ia., OALR
PEELER, C. N.(1920).....6 W. 7th St., Charlotte, N. C., OALR
*PEERY, VANCE PRICE(1929).....Kinston, N. C., ALR
*PEIRCE, HOWARD W.(1923)
704 Med. Arts Bldg., Detroit, Mich., ALR
*PELZ, MORT D.(1929).....Missouri Bldg., St. Louis, Mo., ALR
*PEMBER, AUBREY H.(1926)
500 W. Milwaukee St., Janesville, Wis., OALR
PENDEXTER, RALPH S.(1926)
1424 K St., N. W., Washington, D. C., Op.
PENTECOST, R. S.(1926)....184 Bloor St., Toronto, Canada, OALR
*PETER, L. C.(1914)....1930 Chestnut St., Philadelphia, Pa., Op.
PETERMAN, H. E.(1912)..518 Cathedral St., Baltimore, Md., OALR
PETERSON, R. A.(1920)
150 Fifth Ave., New York City., N. Y., OALR
PFAFFLIN, C. A., 445 Bankers Trust Bldg., Indianapolis, Ind., OALR
PFINGST, A. O.....712 Brown Bldg., Louisville, Ky., Op.
PFINGSTEN, C. F.(1916)
6370 Waterman Ave., St. Louis, Mo., ALR
PHELPS, KENNETH A.(1920)
730 La Salle Bldg., Minneapolis, Minn., OALR
PHILLIPS, W. C.....30 E. 58th St., New York City, N. Y., ALR
PHILLIPS, WM. LINTON..131 Linwood Ave., Buffalo, N. Y., Op.
PHINNEY, F. D.....22 W. 7th St., Cincinnati, O., OALR
PINKERTON, F. J.(1929)
46 Young Hotel Bldg., Honolulu, Hawaii, OALR
PIPER, ARTHUR SYLVESTER(1928).610 S. Monroe, Enid, Okla., OALR
PITKIN, C. E.....Osborn Bldg., Cleveland, O., ALR
*PLUMER, JOHN SHAFFER(1928)
121 University Pl., Pittsburgh, Pa., Op.
*POLLOCK, H. L.(1911).....2551 N. Clark St., Chicago, Ill., ALR
*PONTIUS, P. J.....1831 Chestnut St., Philadelphia, Pa., Op.
*POOS, E. E.(1927).....Fisher Bldg., Detroit, Mich., OALR
POOS, GROVER HEWITT(1928)
University Club Bldg., St. Louis, Mo., OALR
*PORTER, E. H.(1912).....85 Madison St., Tiffin, O., OALR
PORTER, JESSE B.(1920).....415 S. 2nd St., Elkhart, Ind., OALR
PORTER, L. B.(1913).....199 Thayer St., Providence, R. I., OALR
*POST, LAWRENCE T.(1920)
520 Metropolitan Bldg., St. Louis, Mo., Op.
POST, M. HAYWARD(1918)
520 Metropolitan Bldg., St. Louis, Mo., Op.
*POST, WINFRED B.(1921)
First National Bank Bldg., Carthage, Mo., OALR
*POTTER, ALLEN B.(1929).3725 Kingshighway, St. Louis, Mo., ALR
POTTER, GEO. B.(1921)....Medical Arts Bldg., Omaha, Neb., OALR
POTTER, L. F.....68 Walnut St., New Bedford, Mass., OALR
POTTER, PETER(1918).....125 W. Granite St., Butte, Mont., OALR

*Potter, W. W.(1927).....Medical Bldg., Knoxville, Tenn., OALR
Potts, J. B.....Medical Arts Bldg., Omaha, Neb., ALR
*Powell, Lyle Stephenson(1928)
813 Mass. St., Lawrence, Kan., OALR
Powers, Everett(1916)
Central Nat'l. Bank Bldg., Carthage, Mo., OALR
Prangen, Avery de H.(1919), Mayo Clinic, Rochester, Minn., Op.
Pratt, Fred J.(1912)
1753 Medical Arts Bldg., Minneapolis, Minn., OALR
*Pratt, John A.(1910)
1753 Medical Arts Bldg., Minneapolis, Minn., OALR
Prendergast, D. A.....Hanna Bldg., Cleveland, O., OALR
Price, John B.(1929)....513 Swede St., Norristown, Po., ALR
*Price, Norman W.(1908), 445 3rd St., Niagara Falls, N. Y., OALR
Prince, Alexander L.(1928), 36 Pearl St., Hartford, Conn., Op.
*Proetz, A. W.(1923).....Beaumont Bldg., St. Louis, Mo., ALR
*Proud, Willard C.(1920)
Phys. & Surg. Bldg., S. Joseph, Mo., OALR
Prout, A. W.(1920).....370 E. Town St., Columbus, O., OALR
Putnam, F. I.....501 St. Claire Bldg., San Jose, Cal., OALR
*Pyle, Wallace(1915)..15 Exchange Pl., Jersey City, N. J., OALR

Q

Qualls, Harley Winfield(1928)
1291 Union Ave., Memphis, Tenn., OALR
Quinby, Wm. O'G.(1919).....14 James St., Newark, N. J., OALR
*Quincy, J. E.(1927), 270 Commonwealth Ave., Boston, Mass., ALR
*Quittner, S. S.....3912 Prospect Ave., Cleveland, O., OALR

R

Radcliffe, McCluney, 1906 Chestnut St., Philadelphia, Pa., Op.
Ralls, C. T.(1916)....1st Natl. Bank Bldg., Winfield, Kas., OALR
Ralston, Wallace(1918)
1306 Walker Ave., Houston, Tex., Op.
*Ramsey, G. S.(1927), 1496 Mountain St., Montreal, Canada, Op.
Randall, Hiram(1928)....81 Main St., Binghamton, N. Y., ALR
Ravdin, B. D.....Citizens Natl. Bk. Bldg., Evansville, Ind., OALR
*Ravdin, M.....Citizens Natl. Bk. Bldg., Evansville, Ind., OALR
Ray, Victor(1901).....19 W. 7th St., Cincinnati, O., Op.
Reaves, Chas. R.,
Reaves Infir., 117 W. Sycamore St., Greensboro, N. C., OALR
*Reaves, Robert Grisham(1928)
422 W. Cumberland Ave., Knoxville, Tenn., OALR
Reaves, W. P.(1913)
Reaves Infir., 117 W. Sycamore St., Greensboro, N. C., OALR
Rector, A. E.(1915), 112 W. College Ave., Appleton, Wis., OALR
Reeder, J. E.(1916).....Davidson Bldg., Sioux City, Ia., OALR

*Reese, Algernon B.(1928)
50 W. 52nd St., New York City, N. Y., Op.
*Reese, Monroe D.(1926).....123 N. 8th St., Lebanon, Pa., OALR
*Reese, Warren S.(1921)..230 S. 21st St., Philadelphia, Pa., Op.
*Reger, H. S.(1908)....Physicians Bldg., Jamestown, N. Y., OALR
*Reiche, O. C.(1916).....Markle Bk. Bldg., Hazleton, Pa., OALR
Reim, Hugo.....417 Metropolitan Bldg., St. Louis, Mo., Op.
*Reineke, Geo. F.(1926).....Box 137, New Ulm, Minn., OALR
Renaud, G. L.....510 Joseph Campan Ave., Detroit, Mich., OALR
Renner, W. S.(1900)....341 Linwood Ave., Buffalo, N. Y., ALR
*Repass, R. E.(1920)
Lincoln Rd. and Washington Ave., Miami Beach, Fla., ALR
Reuling, F. H.(1924).....Black Bldg., Waterloo, Ia., OALR
Reynolds, H. G.(1909), City Natl. Bk. Bldg., Paducah, Ky., OALR
Reynolds, J. S.(1918)
Medical Arts Bldg., Minneapolis, Minn., OALR
*Rhode, Solon Lafayette(1928)
238 N. 6th St., Reading, Pa., OALR
Rice, Clarence Prentice.....Breckenridge, Minn., OALR
Rice, G. B.(1916).....99 Bay State Road, Boston, Mass., ALR
Rice, John E.(1924).....390 Main St., Worcester, Mass., OALR
Rice, R. H.(1915).....331 Grove St., Milwaukee, Wis., OALR
*Richards, L. G.(1924)..319 Longwood Ave., Boston, Mass., ALR
Richardson, J. J.(1929)
Cook Hosp. and Beall Clinic, Fort Worth, Texas, ALR
*Richardson, S. A.(1925)
111 W. Adams St., Jacksonville, Fla., Op.
Richie, L. T.(1916).....McCormick Bldg., Trinidad, Colo., OALR
Rideout, W. J.....701 State Bk. Bldg., Freeport, Ill., OALR
*Ridpath, Robt. F.,
N. E. cor. 18th and Chestnut Sts., Philadelphia, Pa., LR
Rieger, W. H.(1924).....Hanna Bldg., Cleveland, O., ALR
Rindlaub, Elizabeth P...de Lendrecie Bldg., Fargo, N. D., OALR
Rindlaub, John H.....First Trust Bldg., Pasadena, Cal., OALR
Ringle, Chas. A.(1906)....Coronado Block, Greeley, Colo., ALR
Ringueberg, E. N. S.(1905), 13 Main St., Lockport, N. Y., Op.
Risdon, E. F.(1924).....30 Avenue Rd., Toronto, Canada, ALR
Risser, Edward D.(1928), 310 Choate Bldg., Winona, Minn., OALR
Robb, Mayo(1929).....323 Ellis Bldg., Phoenix, Ariz., ALR
*Roberts, S. E.(1916)
1810 Federal Reserve Bank Bldg., Kansas City, Mo., ALR
Roberts, Wm. H.....65 N. Madison Ave., Pasadena, Cal., Op.
*Robertson, Edw. N.(1918)
132½ W. 6th St., Concordia, Kan., OALR
Robertson, F. D.....Ashton Bldg., Grand Rapids, Mich., OALR
Robinson, H. T.(1916)...Medical Bldg., Cumberland, Md., OALR
Robinson, R. E.(1920), 205½ E. Bremer Ave., Waverly, Ia., OALR
Rogers, A. S.....105 S. Jefferson Ave., Saginaw, Mich., OALR
Rogers, J. T.....Med. Arts Bldg., Montreal, Can., ALR

ROGERS, ROBERT MERRIAM (1928)
680 St. Johns Pl., Brooklyn, N. Y., Op.
ROLLER, LOUIS A. (1920)
446 Paris Ave., S. E., Grand Rapids, Mich., Retired
ROMMEL, JOHN C. . . . . 3701 N. Broad St., Philadelphia, Pa., Op.
ROMONEK, PHILIP L. (1929) . . Med. Arts Bldg., Omaha, Neb., ALR
ROOKER, A. M. (1920), 225 Gluck Bldg., Niagara Falls, N. Y., ALR
ROOST, F. H. (1914) . . . . . Sioux City, Ia., OALR
ROSEBROUGH, F. H. (1929) . . Moore Bldg., San Antonio, Tex., Op.
ROSENBAUM, J., 1396 St. Catherine's St., W., Montreal, Can., Op.
*ROSENBAUM, MORRIS (1929) . 61 W. 88th St., New York, N. Y., Op.
*ROSENBERGER, H. C. (1929) . . . . . Rose Bldg., Cleveland, O., ALR
ROTH, ALBERT W. (1920), Medical Arts Bldg., Tulsa, Okla., OALR
ROTHSCHILD, HAROLD J. . . 239 Lowry Bldg., St. Paul, Minn., OALR
ROTT, O. M. (1915)
Paulsen Medical and Dental Bldg., Spokane, Wash., OALR
ROW, GEO. S. (1911)
906 Hume-Mansur Bldg., Indianapolis, Ind., Op.
ROWLAND, JOHN F. (1911)
Thompson Bldg., Hot Springs, Ark., OALR
*ROWLAND, W. D. (1923) . . 220 Clarendon St., Boston, Mass., Op.
RUBENDALL, C. (1915) . . . . Medical Arts Bldg., Omaha, Neb., OALR
RUBY, F. M. . . . . 1940 E. 3rd Ave., Hibbing, Minn., OALR
RUCKER, C. W. (1929) . . Donaldson Bldg., Minneapolis, Minn., Op.
*RUCKER, E. W., JR. (1916)
926 Woodward Bldg., Birmingham, Ala., ALR
*RUDOLPHY, J. B. (1929) . . . . 1924 Pine St., Philadelphia, Pa., Op.
RUEDEMANN, A. D. (1929)
Cleveland Clinic, Euclid Ave. & 93rd St., Cleveland, O., Op.
*RUNK, R. L. (1929)
Wash. Sanit. & Hosp., Takoma Pk., Washington, D. C., ALR
RUSH, C. C. . . . . 907 U. S. Natl. Bk. Bldg., Johnstown, Pa., OALR
RUSSELL, J. A. M. (1922) . . . . . 813 Sassafras St., Erie, Pa., ALR
*RUSSELL, R. E. (1924) . . 601 Pioneer Bk. Bldg., Waterloo, Ia., OALR
RUST, E. G. . . . . Hanna Bldg., Cleveland, O., Op.A
*RUTHERFORD, C. W. (1925)
University Hospital, Dept. Ophth., Iowa City, Iowa, Op.
RYAN, L. M. (1927) . . . . . Wall Bldg., St. Louis, Mo., ALR
RYCHENER, RALPH ORLANDO (1928)
1720 Exchange Bldg., Memphis, Tenn., Op.
*RYDER, D. R. . . . . 151 Rock St., Fall River, Mass., ALR
*RYERSON, F. L. (1925) . . . . . Fine Arts Bldg., Detroit, Mich., OALR

S

SACCO, ANTHONY (1920)
440 New York Ave., Union City, N. J., OALR
*SALINGER, SAMUEL (1920)
25 E. Washington St., Chicago, Ill., ALR

SALLANDER, F. W.(1919)
385 New Orpheum Bldg., Sioux City, Ia., OALR
SAMUELS, BERNARD(1929) .57 W. 57th St., New York, N. Y., Op.
SANDERS, JOHN GILLIS(1929) .....Park Bldg., Mobile, Ala., ALA
SANDERSON, DAVID D.(1916)
914 Stuart Bldg., Lincoln, Neb., OALR
SANDERSON, H. H.(1906)
David Whitney Bldg., Detroit, Mich., OALR
SANOR, DANIEL GALBREATH, JR.(1928)
206 E. State St., Columbus, O., ALR
SARTAIN, PAUL J.(1921), 2006 Walnut St., Philadelphia, Pa., Op.
SAUER, W. E.(1904) ..Beaumont Med. Bldg., St. Louis, Mo., ALR
*SAUER, W. W.(1910) .....231 Third St., Marietta, O., OALR
SAVAGE, M. M.(1927) .....1720 Eutaw Pl., Baltimore, Md., OALR
*SAVERY, C. E. .....206 Sherland Bldg., South Bend, Ind., OALR
SCAL, JOSEPH COLEMAN(1926)
44 W. 74th St., New York City, N. Y., ALR
SCALES, H. L.(1920) ...Rorabaugh Bldg., Hutchinson, Kas., OALR
*SCALES, J. L.(1915) ....Medical Arts Bldg., Shreveport, La., OALR
SCALES, J. W.(1896) .....204½ Main St., Pine Bluff, Ark., OALR
SCARLETT, HUNTER W.(1919)
230 S. 21st St., Philadelphia, Pa., Op.
*SCHALL, L. A.(1927), 270 Commonwealth Ave., Boston, Mass., Op.
*SCHAPPERT, N. L.(1916)
59 S. Washington St., Wilkes-Barre, Pa., OALR
SCHENCK, C. P.(1920), Med. Arts Bldg., Fort Worth, Tex., OALR
SCHILD, E. H.(1906) .....Brant Bldg., Canton, O., Op.
SCHILLER, A. N.(1918)
249 West End Ave., New York City, N. Y., ALR
SCHLANSER, MAJOR A. E.(1921)
Walter Reed Gen. Hosp., Army Med. Center, Washington, D.C., OALR
SCHLEGEL, HERMAN T.(1916), 501 Third St., Wausau, Wis., OALR
SCHLINDWEIN, G. WM.....138 W. 9th St., Erie, Pa., OALR
SCHLIVEK, KAUFMAN(1925)
1016 Fifth Ave., New York City, N. Y., Op.
SCHNEIDEMAN, T. B.....2045 Chestnut St., Philadelphia, Pa., Op.
*SCHNEIDER, ANTON S.(1928), 56 Court St., Plattsburg, N. Y., Op.A
SCHOLZ, R. P.....Metropolitan Bldg., St. Louis, Mo., ALR
SCHRANT, J. H.(1916)
321 E. 15th St., Hutchinson, Kan., OALR
SCHULZE, E. C.(1926) .....Med. Arts Bldg., Dallas, Tex., OALR
SCHURTER, M. A.(1924) ....114 E. 7th St., Long Beach, Cal., OALR
SCHUSTER, FRANK P.(1927)
403 1st Natl. Bank Bldg., El Paso, Tex., OALR
*SCHUSTER, S. A.(1925) ....1st Natl. Bk. Bldg., El Paso, Tex., ALR
SCHUTZ, W. H. (1921) .....Bryant Bldg., Kansas City, Mo., OALR
SCHWARTZ, A. A.(1927)
941 Park Ave., New York City, N. Y., ALR

SCHWARTZ, FREDERICK O.(1919)
701 Metropolitan Bldg., St. Louis, Mo., Op.
SCHWARTZ, V. J.(1929), Med. Arts Bldg., Minneapolis, Minn., ALR
SCHWARTZ, WILLIAM A.(1928).....Phoenix, Ariz., OALR
*SCHWEINITZ, GEO. E. DE(1905)
1705 Walnut St., Philadelphia, Pa., Op.
SCOTT, J. R.(1920).....201 S. Main St., Ottawa, Kan., OALR
SCOTT, MERLE E.(1926)...1st Natl. Bk. Bldg., Massillon, O., OALR
*SCROGGS, J. J.(1921).....218 E. Third St., Beaver, Pa., Op.A
SEAMAN, GILBERT E.....407 Wells Bldg., Milwaukee, Wis., OALR
*SEARCY, H. B.(1912).....2501 6th St., Tuscaloosa, Ala., OALR
SEAY, D. E.....907 Mercantile Bk. Bldg., Dallas, Tex., OALR
SECOY, F. L.(1922)....301 Davidson Bldg., Sioux City, Ia., OALR
SEDGWICK, O. W.(1917).....Ionia, Mich., OALR
*SEDWICK, W. A.....Republic Bldg., Denver, Colo., Op.
SEEGMAN, SIMON(1918)....3401 5th Ave., Pittsburgh, Pa., ALR
SEELYE, W. K.(1920).....1215 4th Ave., Seattle, Wash., OALR
SELFRIDGE, G. L.....384 Post St., San Francisco, Cal., ALR
SELIGSTEIN, MILTON B.(1926)
801 Union and Planters Bk. Bldg., Memphis, Tenn., OALR
SELLERS, L. M.(1920).....Medical Arts Bldg., Dallas, Tex., ALR
*SENER, WALTER J.....Physicians Bldg., Philadelphia, Pa., Op.
SENSENEY, EUGENE TOWNER(1928)
316 Lister Bldg., St. Louis, Mo., ALR
SEWALL, E. C.(1910)
Stanford Univ. Hospital, San Francisco, Cal., ALR
SEYDELL, E. M.(1916)
1030 First Natl. Bank Bldg., Wichita, Kas., ALR
*SHACKLETON, W. E.(1918), 2323 Prospect Ave., Cleveland, O., Op.
SHAFER, J. J.(1913).....Starks Bldg., Louisville, Ky., OALR
*SHAHAN, W. E.....Metropolitan Bldg., St. Louis, Mo., Op.
SHANKLIN, E. M.....575 Hohman St., Hammond, Ind., OALR
*SHANNON, C. E. G.....1930 Chestnut St., Philadelphia, Pa., Op.
*SHAPERO, I. M.(1919)..289 Alexander St., Rochester, N. Y., OALR
*SHARP, BENJ. S.(1926)....339 Thayer St., Providence, R. I., OALR
SHARRETT, GEO. O.(1921)...119 Bedford, Cumberland, Md., OALR
SHASTID, T. H.(1913), 10th Ave. Med. Bldg., Duluth, Minn., Op.
SHAWHAN, GLENN E.(1913)
Smallwood Bldg., Nampa, Idaho, OALR
*SHEA, J. J.(1919).....1018 Madison Ave., Memphis, Tenn., ALR
SHEAHAN, WM. L.(1926), 59 College St., New Haven, Conn., OALR
SHELLMAN, J. L.(1917)
Lowry Medical Arts Bldg., St. Paul, Minn., OALR
*SHEMELEY, WM. G., JR., 1724 Spruce St., Philadelphia, Pa., OALR
SHERMAN, MAJ. A. ED., M.C., U. S. A.(1913)
Hdqts. Clover Field, Santa Monica, Cal., OALR
*SHERMAN, ELBERT S.(1912)...671 Broad St., Newark, N. J., Op.
*SHIELDS, J. M.(1915).264 Metropolitan Bldg., Denver, Colo., Op.
SHIELDS, LAWRENCE.....Allen Bldg., Xenia, O., ALR

SHIRAS, HOWARD H.(1920)
624 Guardian Bldg., Cleveland, O., Op.
*SHIREY, O. M.(1925).....Osborn Bldg., Cleveland, O., ALR
SHOEMAKER, FERDINAND, U. S. Veterans' Hospital No. 67,
27th and Paseo, Kansas City, Mo., OALR
SHOEMAKER, S. A.....30 E. Church St., Orlando, Fla., OALR
SHOEMAKER, W. A.(1896)
1006 Carleton Bldg., St. Louis, Mo., Op.
*SHREVE, OWEN M.(1905).....162 W. 8th St., Erie, Pa., Op.
SHULTS, E. J. G.(1920)....Haines Bldg., Eldorado, Kansas, OALR
*SHUMAN, G. H.(1917).....Park Bldg., Pittsburgh, Pa., Op.
SHUMATE, D. L.....500 Argyle Bldg., Kansas City, Mo., OALR
*SHURLY, BURT R.....62 Adams Ave., W., Detroit, Mich., ALR
SHUSTER, B. H.(1929)..1930 Chestnut Sa., Philadelphia, Pa., ALR
SHUTE, D. K.....1727 DeSales St., N.W., Washington, D. C., Op.
SIDWELL, C. E.(1929)....Longmont Hosp., Longmont, Colo., ALR
*SIEGEL, FRANCIS X.(1922)
901 Union Central Life Bldg., Cincinnati, O., OALR
*SILBERNAGEL, C. E.(1918)..240 E. State St., Columbus, O., OALR
*SIMON, F. C.(1929).....914 Arcade Bldg., St. Louis, Mo., ALR
*SIMONDS, O. F.....Hanna Bldg., Cleveland, O., Op.
SIMPSON, H. LEE(1921)
1409 David Whitney Bldg., Detroit, Mich., ALR
*SIMPSON, JOHN REID, 825 Highland Bldg., Pittsburgh, Pa., ALR
*SIMPSON, W. LIKELY(1911)
Phys. and Surg. Bldg., Memphis, Tenn., ALR
*SINAMARK, ANDREW(1928), 205 Power Bldg., Hibbing, Minn., OALR
SINGLETON, E. M.(1897)
111 E. Church St., Marshalltown, Ia., OALR
SISSON, RAYMOND J.(1928)
753 David Whitney Bldg., Detroit, Mich., Op.
*SKILLERN, ROSS HALL(1910)
1811 Spruce St., Philadelphia, Pa., R
*SKILLERN, S. R., JR.(1925), 1734 Pine St., Philadelphia, Pa., ALR
SKIRBALL, JOSEPH J.(1927)
353 Commonwealth Ave., Boston, Mass., OALR
SLEIGHT, R. D.(1906)..303 Post Bldg., Battle Creek, Mich., Op.
SLOAN, HENRY L.(1919).....6 W. 7th St., Charlotte, N. C., Op.
SLOCUM, GEO.(1906)
First Nat'l. Bank Bldg., Ann Arbor, Mich., Op.
SMIT, WM. MAURICE(1928)
719 Missouri Theatre Bldg., St. Louis, Mo., ALR
SMITH, ARTHUR E., Donaldson Bldg., Minneapolis, Minn., OALR
*SMITH, CARLIE SOUTER(1927)
214 N. Jefferson Ave., Springfield, Mo., OALR
SMITH, CARROLL L.(1929)
Paulsen Medical and Dental Bldg., Spokane, Wash., OALR
*SMITH, DORLAND(1910), 836 Myrtle Ave., Bridgeport, Conn., Op.A
*SMITH, D. S.(1917).....State Bank Bldg., La Crosse, Wis., OALR

SMITH, E. TERRY.....36 Pearl St., Hartford, Conn., Op.
SMITH, GEO. B.(1914).....Harbin Hospital, Rome, Ga., OALR
SMITH, H. A.(1925)..315 E. Philadelphia St., Whittier, Cal., OALR
SMITH, J. M.....134½ N. Patterson St., Valdosta, Ga., OALR
SMITH, R. C.....Board of Trade Bldg., Superior, Wis., OALR
SMITH, R. N.(1929).....Medical Arts Bldg., Tulsa, Okla., ALR
SMITH, STANLEY.....121 University Pl., Pittsburgh, Pa., Op.
SMITH, VICTOR C.(1912)
1707 Pere Marquette Bldg., New Orleans, La., Op.
SNAPP, CARL FOSTER(1928)
604 Med. Arts Bldg., Grand Rapids, Mich., ALR
SNEED, CARL M.....506 W. Broadway, Columbia, Mo., OALR
SNELL, ALBERT C.(1925)
53 S. Fitzhugh Sa., Rochester, N. Y., Op.
SNYDER, W. H.....220 Michigan St., Toledo, O., OALR
SOMBERG, J. S.(1923).....60 E. 67th St., New York, N. Y., Op.
*SONNENSCHEIN, R.(1915)
180 N. Michigan Ave., Chicago, Ill., ALR
*SPAETH, EDMUND B.(1928)
1930 Chestnut St., Philadelphia, Pa., Op.
SPAKE, LAVERNE B.(1920)
322 Brotherhood Bldg., Kansas City, Kans., ALR
SPALDING, J. A.(1896).....627 Congress St., Portland, Me., Op.A
*SPENCER, FRANK R.(1904), Physicians Bldg., Boulder, Colo., OALR
SPENCER, GEO. ALBERT(1928)
1127 Eleventh St., Sacramento, Cal., OALR
SPENCER, S. C.(1929)....605 Columbus, Ave., Waco, Texas, Op.
SPENCER, W. H...1830 S. Rittenhouse Sq., Philadelphia, Pa., ALR
SPENGLER, JOHN ARTHUR(1916)
423 S. Main St., Geneva, N. Y., Op.
SPIESMAN, I. G.(1929)..25 E. Washington St., Chicago, Ill., ALR
SPITZE, E. C.(1916).....Murphy Bldg., East St. Louis, Ill., Op.
SPROULE, RALPH P.(1928)
309 Goldsmith Bldg., Milwaukee, Wis., OALR
*STAHLMAN, FRED C.....Commercial Bldg., Wavnesburg, Pa., Op.
STANBERRY, HENRY.....556 Doctors Bldg., Cincinnati, O., Op.
STANFORD, J. B.....1052 Madison Ave., Memphis, Tenn., OALR
STANTON, HARMON LAMONTTE(1928)
Cleveland Life Bldg., Evansville, Ind., OALR
STARK, JESSE B.(1925), 45 Park Ave., New Your City, N. Y., Op.
STARK, WM. B.(1927).....Mayo Clinic, Rochester, Minn., ALR
*STAUFFER, FRED(1912)
Medical Arts Bldg., Salt Lake City, Utah, OALR
*STEELE, WILLARD(1928), Provident Bldg., Chattanooga, Tenn., OALR
*STEGMAN, LOUIE V.(1919)..Sanitarium, Battle Creek, Mich., Op.
STEIM, J. M.(1914)
First Natl. Bank Bldg., New Kensington, Pa., Op.
STEIN, O. J.....30 N. Michigan Blvd., Chicago, Ill., ALR
STEINFELD, A. L.(1922).....125 15th St., Toledo, O., OALR

STEVENSON, D. W.....165 E. Market St., Akron, O., OALR
STEVENSON, WALTER (1915)....W. C. U. Bldg., Quincy, Ill., OALR
STEWART, WALTER E. (1920)
Tribune Bldg., Terre Haute, Ind., OALR
*STIEREN, E. (1905)..626 Union Trust Bldg., Pittsburgh, Pa., Op.
*STILES, CHAS. M. (1908)..1130 Spruce St., Philadelphia, Pa., Op.
STILLSON, H.....Seaboard Bldg., Seattle, Wash., OALR
STIMSON, GEORGE W. (1919)....Packard Bldg., Warren, O., OALR
STINSON, WM. DAVID (1928)
805 Medical Arts Bldg., Memphis, Tenn., ALR
STITT, H. L. (1927).....656 Doctors Bldg., Cincinnati, O., OALR
STOCKLER, J. A. (1917)..332 North Fifth St., Reading, Pa., OALR
STOKES, WM. H. (1926)....4105 Live Oak St., Dallas, Tex., Op.
STOTTER, ARTHUR L. (1916), 1148 Euclid Ave., Cleveland, O., OALR
*STOUT, PHILIP S.....269 S. 19th St., Philadelphia, Pa., ALR
STOUTENBOROUGH, WM. A. (1927)
318 East State St., Columbus, O., OALR
STRADER, GEO. L. (1904)..408 Hynds Bldg., Cheyenne, Wyo., OALR
*STRAUSS, J. F. (1925)....104 S. Michigan Blvd., Chicago, Ill., ALR
STRICKLER, D. A.....Republic Bldg., Denver, Colo., Op.
STRONG, J. C. (1915)....810 Harrison Ave., Leadville, Colo., OALR
STROUD, EDWARD FRANK (1926)
408 City Natl. Bank Bldg., Corpus Christi, Tex., ALR
STROUSE, FREDERICK M. (1920)
1301 Spruce St., Philadelphia, Pa., ALR
STROUT, E. S. (1907)....74 S. 11th St., Minneapolis, Minn., OALR
STROUT, G. E. (1921)..1009 Nicollet Ave., Minneapolis, Min., OALR
STUCKY, J. A. (1896)....Fayette Bk. Bldg., Lexington, Kv., OALR
STUEBER F. G. (1905).....209 W. North St., Lima, O., OALR
STUEBER, PAUL J. (1917).....209 W. North St., Lima, O., OALR
STURGES, LEIGH F. (1920)
121 E. 60th St., New York City, N. Y., ALR
STURM, S. A. (1916)
7036 Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
*SUKER, GEO. F. (1896)...25 E. Washington St., Chicago, Ill., Op.
*SULLIVAN, J. J., JR. (1923)
228 Scranton Life Bldg., Scranton, Pa., ALR
SULZER, G. A. (1907).....327 E. State St., Columbus, O., OALR
SULZMAN, FRANK MALCOLM (1922)
1831 Fifth Ave., Troy, N. Y., OALR
*SUMMERS, WM. S. (1927)
1613 David Whitney Bldg., Detroit, Mich., OALR
SUTHERLAND, J. M. (1929)
8-257 General Motors Bldg., Detroit, Mich., ALR
SWAB, CHARLES MARION (1928)
Medical Arts Bldg., Omaha, Neb., Op.
SWAN, C. J. (1911).....636 Church St., Evanston, Ill., OALR
SWEET, ROBERT B. (1926)
Pacific S. W. Bk. Bldg., Long Beach, Cal., OALR

SWERDFEGER, E. B.(1927), 1008 Republic Bldg., Denver, Colo., ALR
SWETNAM, C. K.(1915)....Masonic Temple, Prescott, Ariz., ALR

T

TANGEMAN, H. F.(1919)
606 Union Central Bldg., Cincinnati, O., ALR
TAQUINO, GEORGE J.(1920)
208 Audubon Bldg., New Orleans, La., ALR
TARUN, WILLIAM....104 West Madison St., Baltimore, Md., OALR
*TASSMAN, ISAAC C.(1929)
Medical Arts Bldg., Philadelphia, Pa., Op.
TAYLOR, CHARLES B.(1921)....118 E. 2nd St., Ottumwa, Ia., ALR
TAYLOR, LILLIAN E.(1905)
25 E. Washington St., Chicago, Ill., ALR
TAYLOR, M. C.(1915).....1204 May Bldg., Pittsburgh, Pa., Op.
TEALL, RAYMOND EDWIN(1928)
1100 Rialto Bldg., Kansas City, Mo., ALR
*THEOBALD, GEORGIANA DVORAK(1929)
1800 S. Ashland Ave., Chicago, Ill., Op.
*THEOBALD, JOHN J.(1929)
58 E. Washington St., Chicago, Ill., ALR
THIGPEN, C. A.....401 So. Court St., Montgomery, Ala., OALR
THOMAS, C. D.(1914), Central Natl. Bank Bldg., Peoria, Ill., OALR
THOMAS, FRANCIS W.(1916), 327 E. State St., Columbus, O., OALR
THOMAS, H. G.(1926).....411 30th St., Oakland, Cal., OALR
THOMAS, JAMES W.(1928)..1108 Sharp Bldg., Lincoln, Neb., OALR
THOMASON, H. E.....Rialto Bldg., Kansas City, Mo., ALR
THOMASSON, W. J.(1910).....942 York St., Newport, Ky., ALR
THOMPSON, E. H.....8 W. 9th St., Cincinnati, O., OALR
*THOMPSON, G. H.(1909)....74 North St., Pittsfield, Mass., OALR
THOMPSON, HOWARD E.(1928), 1100 Main St., Dubuque, Ia., OALR
THOMPSON, PETER H.(1914)
308 Commonwealth Ave., Boston, Mass., Op.
THOMPSON, J. J.(1907)
531 East Lincoln Ave., Mt. Vernon, N. Y., ALR
TILDERQUIST, D. L.(1925), 425 Bradley Bldg., Duluth, Minn., OALR
TIMBERMAN, ANDREW.....Beggs Bldg., Columbus, O., OALR
TINDOLPH, L. W.....1st Natl. Bank Bldg., Olean, N. Y., ALR
*TINGLEY, LOUISA PAINE(1913)
9 Massachusetts Ave., Boston, Mass., Op.
Also 171 Westminster St., Providence, R. I.
TITSWORTH, GUY.....111 W. 4th St., Sedalia, Mo., OALR
TIVNEN, RICHARD J.....30 N. Michigan Ave., Chicago, Ill., OALR
TOBEY, GEO. L., JR...270 Commonwealth Ave., Boston, Mass., ALR
TODD, GROVER CLEVELAND(1922)
307 Westinghouse Bldg., Pittsburgh, Pa., ALR
*TOMASSENE, R. A.(1922)
Wheeling Bank & Trust Bldg., Wheeling, W. Va., OALR

*Tomlin, W. S.(1912)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
Toms, S. W. S.(1909)....120 S. Broadway, Nyack, N. Y., OALR
Tooker, C. W.(1907).....Carleton Bldg., St. Louis, Mo., Op.
Townsend, John Ferrars(1923)
85 Wentworth St., Charleston, S. C., OALR
Trible, G. B.(1920)
1801 Eye St., N. W., Washington, D. C., ALR
Trimble, Clarence S.(1920)
405 Citizens Natl. Bank Bldg., Emporia, Kan., OALR
Tripp, Ira A.(1920).....436 Rose Bldg., Cleveland, O., OALR
Tripp, Leroy R.....306 Security Bldg., Sioux City, Ia., OALR
Tucker, Gabriel(1923)
Graduate Hospital, 19th & Lombard St., Philadelphia, Pa., B
Tucker, James C.(1920)..515½ Court St., Beatrice, Neb., OALR
Tuckerman, W. C.(1911)..733 Osborn Bldg., Cleveland, O., Op.
*Tuckerman, W. H.....733 Osborn Bldg., Cleveland, O., OALR
Turnbull, F. M.(1916)
806 Pacific Mutual Bldg., Los Angeles, Cal., ALR
*Turner, H. H.(1914), 902 Professional Bldg., Pittsburgh, Pa., Op.
Turner, Oliver W.(1909). Masonic Temple, Augusta, Me., OALR

U

*Upham, Helen F.(1907), 305 3rd Ave., Asbury Park, N. J., OALR
Uren, C. T.(1919), 537 City Natl. Bank Bldg., Omaha, Neb., ALR
*Urner, M. H.....2600 Union Central Bldg., Cincinnati, O., OALR

V

Vail, Derrick T., Sr.....24 E. 8th St., Cincinnati, O., Op.
Vail, H. H.(1924).....24 E. 8th St., Cincinnati, O., ALR
Van Kirk, Frank J.(1920)
Bellingham Natl. Bank Bldg., Bellingham, Wash., OALR
Vankirk, V. E.....626 Union Trust Bldg., Pittsburgh, Pa., Op.
Van Meter, Eugene R.(1927)
4473 Washington Ave., St. Louis, Mo., ALR
Van Poole, G. McD.(1925)
Young Bldg., Honolulu, T. Hawaii, OALR
Vaughan, G. E.....711 3rd St., Louisville, Ky., OALR
Vaughn, E. H.....Citizens Natl. Bank, Tyler, Tex., OALR
Veasey, C. A., Sr.(1905)
Paulsen Medical and Dental Bldg., Spokane, Wash., OALR
Veassy, C. A., Jr.(1927)
404 Paulsen Medical and Dental Bldg., Spokane, Wash., OALR
Vinsonhaler, F.(1902), Urquhart Bldg., Little Rock, Ark., OALR
Voigt, C. B.(1912).....1702 Broadway, Mattoon, Ill., OALR

VON COLDITZ, G. THOMSEN
25 E. Washington St., Chicago, Ill., OALR
*VON DER HEYDT, ROBERT (1926)
25 E. Washington St., Chicago, Ill., Op.

W

WAGENER, HENRY P. (1926). Mayo Clinic, Rochester, Minn., Op.
*WAGERS, ARTHUR J. (1920)
1429 Spruce St., Philadelphia, Pa., ALR
WAGNER, CARL B. (1912) . . . . 31 North State St., Chicago, Ill., OALR
WAGNER, W. A. (1927)
1003 Pere Marquette Bldg., New Orleans, La., ALR
WALKER, CHAS. C. (1920), 604 Maccabee Bldg., Detroit, Mich., Op.
WALKER, C. E. (1920) . . . . 517 Jacobson Bldg., Dener, Colo., Op.
WALKER, GEO. H. (1927) . . . . . 172 Main St., Winona, Minn., OALR
WALKER, HERBERT S. . . . . 30 N. Michigan Ave., Chicago, Ill., OALR
WALKER, WALDO WEBSTER (1928)
99 Bay State Road, Boston, Mass., ALR
WALSH, JOSEPH MARK (1922) . . . . . Rapid City, S. D., OALR
WARNER, ROBT. J. (1929) . . . . Doctors Bldg., Nashville, Tenn., Op.
*WARREN, WILLIAM C., JR. (1928)
618 Atlanta Natl. Bank Bdlg., Atlanta, Ga., ALR
WARWICK, HAROLD LYNWOOD (1928)
913 Med. Arts Bldg., Fort Worth, Tex., ALR
WATKINS, EDWIN DIAL (1926)
1412 Exchange Bldg., Memphis, Tenn., OALR
WATSON, H. D. (1915) . . . . 151 Front St., Binghamton, N. Y., OALR
WATSON, JAMES A. . . . . 74 S. 11th St., Minneapolis, Minn., OALR
*WATSON, R. S. (1923) . . . . . Graebrier Bldg., Saginaw, Mich., OALR
*WAUGH, J. M.,
Cleveland Clinic, Euclid Ave. at 93rd St., Cleveland, O., OALR
*WEAVER, JOHN O. (1929) . 608 Sheridan Ave., Shenandoah, Ia., ALR
*WEAVER, T. W. (1914) . . 201½ N. Main St., Wichita, Kan., Op.
WEBER, HENRY CHARLES (1928)
1346 Chestnut St., Waukegan, Ill., OALR
*WEED, HARRY M. (1908) . . 196 Linwood Ave., Buffalo, N. Y., Op.
WEEKS, JOHN E. . . Dunthorpe Rt. 8, Portland, Ore., Retired, Op.
*WEEKS, WEBB W. (1923)
20 E. 53rd St., New York City, N. Y., Op.
*WEIH, E. P. (1920) . . . . . Wilson Bldg., Clinton, Ia., OALR
WEIL, A. I. . . . . 3503 Prytania St., New Orleans, La., ALR
*WEINBERGER, NELSON S. (1928)
Robert Packer Hospital, Savre, Pa., OALR
WEINSTEIN, M. A. (1927), 1309 Spruce St., Philadelphia, Pa., ALR
WEISSER, E. A. . . . . 806 May Bldg., Pittsburgh, Pa., Op.
WELCH, ROBERT S. G. (1921)
46 State St. Circle, Annapolis, Md., OALR
*WELLS, DAVID W. (1908), Hotel Westminster, Boston, Mass., Op.

WELLS, GEO. S.....8 E. Arrellaga, Santa Barbara, Cal., ALR
WELTY, C. F.(1926).....210 Post St., San Francisco, Cal., ALR
*WENCKE, CARL G., Battle Creek Sanit., Battle Creek, Mich., ALR
*WENDEL, J. S.(1922)
744 David Whitney Bldg., Detroit, Mich., ALR
WESSELS, ANDREW B.(1929)
Medical-Dental Bldg., San Diego, Cal., ALR
WESTLAKE, SAMUEL B., Beaumont Med. Bldg., St. Louis, Mo., ALR
WETHERBEE, CHARLES(1926)
Booth Bldg., Twin Falls, Idaho, OALR
*WEYMANN, M. F.(1929)
2007 Wilshire Blvd., Los Angeles, Cal., Op.
WHALEN, EDWARD JOSEPH(1928)
750 Main St., Hartford, Conn., ALR
WHELAN, G. L.(1925)....2100 Walnut St., Phildelphia, Pa., ALR
WHEELER, ERNEST C.....Townsend Bldg., Tacoma, Wash., OALR
*WHEELER, J. M.(1920), 30 W. 59th St., New York, N. Y., Op.
WHEELER, MERRITT W...1027 Lowry Bldg., St. Paul, Minn., OALR
*WHERRY, W. P.(1910), 1500 Med. Arts Bldg., Omaha, Neb., OALR
WHISNANT, A. M...517 Professional Bldg., Charlotte, N. C., OALR
WHITAKER, J.(1917).....905 Congress Bldg., Miami, Fla., OALR
WHITE, CHAS. P...507 Citizens Bk. Bldg., Wilmington, Del., OALR
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1610 Sherbrooke St., W. Montreal, Can., ALR
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813 Gurley Bldg., Stamford, Conn., OALR
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*WIENER, MEYER.....900 Carleton Bldg., St. Louis, Mo., Op.
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WILBUR, EDWARD P.
Kalamazoo Trust & Sav. Bk. Bldg., Kalamazoo, Mich., OALR
*WILDER, WM. H.(1896), 122 S. Michigan Blvd., Chicago, Ill., Op.
WILLIAMS, HORACE J.(1922)
5908 Green St., Germantown, Philadelphia, Pa., ALR
*WILLIAMS, M. M.(1927)
Medical Arts Bldg., Scranton, Pa., Op.
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WILLIAMS, WALTON H.(1921), Castell Bldg., Middletown, O., OALR
WILLSON, WESLEY W.(1928)
2204 Eaton Tower, Detroit, Mich., OALR
WINDHAM, ROBERT EDWARD(1928)
234 W. Beauregard Blvd., San Angelo, Texas, OALR
WINGER, IRA B.(1927).....225 Michigan St., Toledo, O., OALR
*WINTER, GEORGE E.(1921)
409 McBride Bldg., Jackson, Mich., OALR
*WINTER, JOHN A.(1923)....205 W. 2nd St., Duluth, Minn., OALR
WISE, RALPH C.(1914).....News Bldg., Mansfield, O., OALR
WOLD, K. C(1924)
1027 Lowry Med. Arts Bldg., St. Paul, Minn., OALR

WOLF, SAMUEL(1929)...55 E. Washington St., Chicago, Ill., Op.
*WOLFE, CLAUDE T.(1916)...Heyburn Bldg., Louisville, Ky., OALR
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415 Masonic Temple, Marshalltown, Ia., OALR
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Medical Arts Bldg., Minneapolis, Minn., OALR
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201 Beaumont Medical Bldg., St. Louis, Mo., ALR
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421 Huguenot St., New Rochelle, N. Y., ALR
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Woodson Eye, Ear, Nose & Throat Hosp., Temple, Tex., OALR
*WOODSON, J. M.(1914)
Cor. Avenue G and Fifth, Temple, Tex., OALR
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1424 Maccabees Bldg., Detroit, Mich., OALR
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307 Yeates Bldg., Minneapolis, Minn., OALR
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143 College St., Toronto, Can., Op.
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Peoples Bk. Bldg., Rochester, Pa., Op.
Also Westinghouse Bldg., Pittsburgh, Pa.,
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Shenandoah Life Bldg., Roanoke, Va., Op.
*YOUNG, H. B.(1897)....204 Jefferson St., Burlington, Iowa, OALR

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*ZEHNDER, A. CHARLES(1919)
188 Roseville Ave., Newark, N. J., Op.
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*ZIEGELMAN, ED. F.(1929).....273 B St., San Meteo, Cal., ALR
Also 384 Post St., San Francisco, Cal.
*ZINKHAN, A. M.(1920), The Rochambeau, Washington, D. C., ALR
*ZUBAK, MATTHEW FRANCIS
Citizens Mutual Trust Bldg., Wheeling, W. Va., OALR

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BARKAN, OTTO.....490 Post St., Op.
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DOLMAN, PERCIVAL.....490 Post St., Op.
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GRVEN, L. D.....The Green's Eye Hospital, Op.
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IRVINE, ROBERT STEELE.....490 Post St., ALR
MCCOOL, J. L.....450 Sutter St., Op.
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WELTY, C. F.....210 Post St., ALR
ZIEGELMAN, EDWARD FRANK.....384 Post St., ALR

SAN JOSE

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WELS, GEO. S.....8 E. Arrellaga, ALR

SAN METEO

ZIEGELMAN, EDWARD FRANK.....273 B St., ALR

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BANE, WM. M.....1005 Republic Bldg., OALR
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BOYD, E. T.....823 Majestic Bldg., OALR
BROWN, MORROW D.....432 Republic Bldg., ALR
CARMODY, T. E.....Metropolitan Bldg., ALR
COLLINS, E. W.....508 Majestic Bldg., ALR
COOPER, C. E.....652 Metropolitan Bldg., ALR
CRISP, WM. H.....Metropolitan Bldg., Op.
DARROW, C. H.....110 Metropolitan Bldg., ALR
FINNOFF, W. C.....217 Imperial Bldg., Op.
FOLEY, J. W.....Majestic Bldg., OALR
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JACKSON, EDWARD.....314 14th St., Op.
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LEYDA, JAMES H.....Metropolitan Bldg., ALR
LOCHARD, L. B.....Metropolitan Bldg., ALR
MCCAW, JOHN A.....418 Majestic Bldg., Op.
MCKEOWN, ELMER F.....Metropolitan Bldg., OALR
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O'ROURKE, D. H.....217 Imperial Bldg., Op.
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SHIELDS, J. M.....264 Metropolitan Bldg., Op.
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SWERDFEGER, E. B.....1008 Republic Bldg., ALR
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CHICAGO HEIGHTS

GORRELL, T. J. H.....16th and Vincennes Ave., OALR

DANVILLE

CARMODY, THOMAS J.....408 The Temple Bldg., OALR

DIXON

MCNICHOLS, W. A.....Dixon Natl. Bank Bldg., OALR

EAST ST. LOUIS

HANSON, WM. LUDWIG.....318 Murphy Bdlg., ALR
SPITZE, E. C.....Murphy Bldg., Op.

EVANSTON

BOOT, G. W.....811 Sherman Ave., OALR
DODD, OSCAR.....636 Church St., Op.
GALLOWAY, T. C.....636 Church St., ALR
SWAN, C. J.....636 Church St., OALR

FREEPORT

CLARK, J. SHELDON .....509 State Bank Bldg., OALR
RIDEOUT, W. J.....701 State Bank Bldg., OALR

GALESBURG

MATHENY, R. C.....405 Bk. of Galesburg Bldg., OALR

HOOPESTON

EAREL, A. M.....Willdon Bldg., OALR

HUBBARD WOODS

LOEB, CLARENCE.....1054 Tower Road., Op.

JACKSONVILLE

ADAMS, A. L.....Professional Bldg., OALR

JOLIET

WOODRUFF, H. W.....500 N. Ottawa St., OALR

KANKAKEE

GEIGER, C. W.....Arcade Bldg., OALR

LA SALLE

WOODS, R. H.....Neustadt Bldg., Op.

MACOMB

DUNTLEY, G. S.....103 So. Side Sq., OALR

MATTOON

VOIGT, C. B.....1702 Broadway, OALR

MOLINE

FLATLEY, T. J.....Reliance Bldg., OALR

PEKIN

GLASFORD, S. T.....Box 167, OALR

PEORIA

BLICKENSTAFF, A. J.....Peoria Life Bldg., OALR
BROBST, C. H.....Central Bank Bldg., OALR
DUANE, J. F.....Jefferson Bldg., OALR
FLOYD, T. W.....811 Peoria Life Bldg., OALR
THOMAS, C. D.....Central National Bank Bldg., OALR
WILLIAMS, W. C.....Jefferson Bldg., OALR

PONTIAC

MIDDLETON, A. B.....Sterry Blk., OALR

QUINCY

MERCER, RAY.....W. C. U. Bldg., OALR
STEVENSON, WALTER.....W. C. U. Bldg., OALR

ROCKFORD

FRINGER, W. R.....William Brown Bldg., Op.
WORMLEY, H. R.....Talcott Bldg., OALR

SPRINGFIELD

DEAL, JOHN FRANCIS.....1st Natl. Bk. Bldg., OALR
HAGLER, E. E.....401 East Capitol Ave., OALR

STERLING

BRODRICK, FRANK W.....Central Trust Bldg., OALR

STREATOR

LESTER, HARRY S.....420 Murray Bldg., OALR

WAUKEGAN

WEBER, HENRY CHARLES.....1346 Chestnut, OALR

INDIANA

ELKHART

PORTER, JESSE B.....415 S. 2nd St., OALR

EVANSVILLE

BROSE, L. D.....501 S. East First St., OALR
DYER, WALLACE CURTIS.....206 Cleveland Life Bldg., ALR
KNAPP, B.....Cleveland Life Bldg., OALR
RAVDIN, B. D.....Citizens Nat'l Bk. Bldg., OALR
RAVDIN, M.....Citizens Nat'l Bk. Bldg., OALR
STANTON, HARMON LAM.....Cleveland Life Bldg., OALR

FORT WAYNE

BARTHOLOMEW, A. C.....1020 Harrison St., OALR
BULSON, A. E.....406 W. Berry St., OALR
BULSON, EUGENE LORING.....406 W. Berry St., OALR
MCBRIDE, W. O.....2902 S. Fairchild Ave., OALR

HAMMOND

KUHN, HUGH A.....1st Trust Bldg., ALR
LLOYD, A. W.....804 Lloyd Bldg., OALR
SHANKLIN, E. M.....575 Hohman St., OALR

INDIANAPOLIS

ADAMS, D. S. .....508 Hume-Mansur Bldg., OALR
ARONSON, SIDNEY S.....Hume-Mansur Bldg., ALR
BERRY, D. F.....Hume-Mansur Bldg., ALR
CARMACK, J. W.....505 Hume-Mansur Bldg., ALR
CHAPPELL, R. S.....110 N. Illinois St., ALR
CLEVENGER, W. F.....Hume-Mansur Bldg., ALR
CRAFT, KENNETH L.....1003 Hume-Mansur Bldg., ALR
DEWEY, C. H., MAJ., M.C.,
U. S. Army Hospital, Ft. Benjamin Harrison, OALR
HUGHES, W. F.....401 Hume-Mansur Bldg., Op.
KEARBY, D. O.....709 Hume-Mansur Bldg., ALR
LARKIN, BERNARD J.....Hume-Mansur Bldg., Op.
LINGEMAN, EDWARD LAUREL.....406 Pennway Bldg., ALR
MCCASKEY, C. H.....Continental Bk. Bldg., ALR
MASTERS, ROBERT J.....23 E. Ohio St., Op.
MOLT, WM. F.....529 Bankers Trust Bldg., ALR
NEWCOMB, JOHN R.....410 Hume-Mansur Bldg., Op.
OVERMAN, F. V.....Hume-Mansur Bldg., ALR
PFAFFLIN, C. A.....445 Banker Trust Bldg., OALR

ROW, GEO. S.....906 Hume-Mansur Bldg., Op.
TOMLIN, W. S.....Hume-Mansur Bldg., ALR
WRIGHT, J. W. M.....Hume-Mansur Bldg., ALR

KOKOMO

ADAMS, CHAS. JOSEPH.....618 A. & L. Bldg., OALR

LOGANSPORT

EGAN, BURTON WRIGHT.....408 North St., OALR

MARION

MILLER, C. E.....Western Reserve Bldg., OALR
BRAUNLIN, WM. H.....Marion Nat'l Bank Bldg., OALR

MICHIGAN CITY

KRIEGER, G. M.....701 Washington St., OALR

MUNCIE

MILLER, C. E.....Western Reserve Bldg., OALR

SOUTH BEND

BOONE, J. C.....213 Pythian Bldg., ALR
BOYD-SNEE, HARRY.....716 J. M. S. Bldg., ALR
CASSADY, J. VERNAL.....654 Associate Bldg., OALR
EDWARDS, SLOCOMB R.....Christman Bldg., OALR
LENT, E. J.....122 N. Lafayette Blvd., OALR
SAVERY, C. E.....206 Sherland Bldg., OALR

TERRE HAUTE

ALLEN, ORRIS T.....422 Rose Dispensary, OALR
GILLUM, J. R.....221 S. 6th St., OALR
STEWART, WALTER E.....Tribune Bldg., OALR

WARSAW

HOWARD, C. N.....210 S. Indiana St., OALR

IOWA

AMES

DYER, BENJAMIN G.....ALR

BURLINGTON

LA FORCE, E. F.....Iowa State Bk. Bldg., OALR
YOUNG, H. B.....204 Jefferson St., OALR

CEDAR RAPIDS

BAILEY, FRED W.....Merchants Natl. Bk. Bldg., OALR
IVINS, H. M.....815 Merchants Natl. Bk. Bldg., OALR

CLINTON

WEIH, E. P.....Wilson Bldg., OALR

COUNCIL BLUFFS

DEAN, F. W.....532 First Ave., OALR
HOWARD, LLOYD G.....Council Bluffs Clinic, OALR
MAIDEN, S. D.....403 Bennett Bldg., OALR

CRESTON

CHILDS, H. A.....Eagle Blk., OALR

DAVENPORT

HANDS, S. GILLOTT.....602 1st Natl. Bk. Bldg., ALR
HARKNESS, GORDON F.....Putnam Bldg., OALR
HOFMANN, W. P.....707 Kahl Bldg., OALR
LAMB, HARRY HOWE.....American Bank Bldg., ALR

DES MOINES

JONES, CECIL C.....1120 Equitable Bldg., OALR
PEARSON, WM. W.....417 Bankers Trust Bldg., OALR

DUBUQUE

THOMPSON, HOWARD E.....1100 Main St., OALR

FORT DODGE

CHASE, S. B.....Carver Bldg., OALR

INDEPENDENCE

AGNEW, F. F.....120 Third Ave., S. E., OALR

IOWA CITY

BOILER, W. F.....Dey Bldg., OALR
LIERLE, DEAN McA.....University Hospital, ALR
O'BRIEN, C. S.....University Hospital, Op.
RUTHERFORD, C. W.....Univ. Hosp., Dept. Ophth., Op.

KEOKUK

LAPSLEY, ROBERT M.....Masonic Temple, OALR

MARSHALLTOWN

COBB, EDWIN.....Masonic Temple Bldg., OALR
FRENCH, R. F.....Masonic Temple Bldg., OALR
SINGLETON, E. M.....111 E. Church St., R
WOLFE, OTIS R.....415 Masonic Temple, OALR

MASON CITY

FALLOWS, HOWARD D.....Park Hospital Clinic, OALR
O'BRIEN, STEVE A.....First Natl. Bk. Bldg., OALR

MUSCATINE

CONE, ALFRED JARED.....404 Laurel Bldg., ALR
JOHNSTON, W. H.....508 Laurel Bldg., OALR

NEWTON

ENGLE, H. P.....Maytag Bldg., OALR

OTTUMWA

TAYLOR, CHARLES B.....118 E. 2nd St., ALR

SHENANDOAH

WEAVER, JOHN O.....608 Sheridan Ave., ALR

SIOUX CITY

DECKER, J. C.....Francis Bldg., OALR
FRANCHERE, FRED'K. E.....Farmers Loan & Trust Bldg., OALR
GITTINS, T. R.....401 Davidson Bldg., OALR

NAFTZGER, J. B.....Davidson Bldg., OALR
REEDER, J. E.....Davidson Bldg., OALR
ROOST, F. H.....OALR
SALLANDER, F. W.....New Orpheum Bldg., OALR
SECOY, F. L.....301 Davidson Bldg., OALR
TRIPP, LEROY R.....306 Security Bldg., OALR

WATERLOO

JOYNT, A. J.....Black Bldg., OALR
REULING, F. H.....Black Bldg., OALR
RUSSELL, R. E.....601 Pioneer Bk. Bldg., OALR

WAVERLY

ROBINSON, R. E.....205½ E. Bremer Ave., OALR

KANSAS

ATCHISON

BRIBACH, EUGENE J.....212 Simpson Bldg., OALR

COFFEYVILLE

CHADWICK, IRA B.....4 Terminal Bldg., Op.

CONCORDIA

ROBERTSON, EDW. N.....132½ W. 6th St., OALR

EL DORADO

SHULTS, E. J. G.....Haines Bldg., OALR

EMPORIA

GRANGER, W. B.....City Natl. Bk. Bldg., OALR
TRIMBLE, CLARENCE S.....405 City Natl. Bk. Bldg., OALR

HUTCHINSON

GREEVER, B. L.....610 Rorabaugh-Wiley Bldg., OALR
SCALES, H. L.....Rorabaugh-Wiley Bldg., OALR
SCHRANT, J. H.....321 E. 15th St., OALR

KANSAS CITY

LIDIKAY, C. J.....903 N. 7th St., OALR
MCCARTNEY, OLIVER P.....Huron Bldg., OALR
SPAKE, LAVERNE B.....322 Brotherhood Bldg., ALR

LAWRENCE

POWELL, LYLE S.....813 Mass. St., OALR

LEAVENWORTH

HAAS, FREDERICK J.....Masonic Bldg., OALR

OTTAWA

SCOTT, J. R.....201 S. Main St., OALR

PARSONS

LANDES, GEORGE A.....OALR

PRATT

JENKINS, MIKE CLEMONT.....223 S. Main St., ALR

TOPEKA

ALKIRE, H. L.....614 Kansas Ave., OALR
ALLEN, G. HENRY.....Mills Bldg., OALR
ALLEN, GEO. V.....835 Kansas Ave., OALR

WICHITA

BROWNELL, MORTON EMMONS.....1019 First Natl. Bk. Bldg., Op.
CHENEY, JAMES W.....702 Schweiter Bldg., OALR
GILLETT, WILBUR G.....201½ N. Main St., Op.
GSELL, J. F.....911 Beacon Bldg., OALR
MAGGARD, D. I.....715 Orpheum Bldg., OALR
PALMER, EDW. M.....629 Beacon Bldg., OALR
SEYDELL, E. M.....1030 First Natl. Bk. Bldg., ALR
WEAVER, T. W.....201½ N. Main St., Op.

WINFIELD

RALLS, C. T.....First National Bank Bldg., OALR

KENTUCKY

BEREA

COWLEY, ROBERT HENRY.....OALR

COVINGTON

BLEDSOE, R. W.....1005 Madison Ave., OALR

FULTON

COHN, SELDON.....OALR

HARLAN

GUNN, MILUS LYDELL.....Smith Bldg., ALR

LEXINGTON

ARMSTRONG, R. M.....1010 Fayette Bk. Bldg., OALR
DEWEESE, CLARENCE.....201 West Second St., OALR
MARKS, SAMUEL BLACKBURN.....Security Trust Bldg., ALR
OFFUTT, W. N.....406 Security Trust Bldg., OALR
STUCKY, J. A.....Fayette Bk. Bldg., OALR

LOUISVILLE

BASS, A. L.....738 Francis Bldg., OALR
DABNEY, S. G.....Starks Bldg., OALR
DEAN, WALTER.....300 Francis Bldg., OALR
DULANEY, OCTAVUS.....811 Brown Bldg., ALR
HALL, G. C.....705 Brown Bldg., OALR
HEITGER, J. D.....808 Francis Bldg., OALR
PFINGST, A. O.....712 Brown Bldg., Op.
SHAFER, J. J.....Starks Bldg., OALR
VAUGHAN, G. E.....711 Third St., OALR
WOLFE, CLAUDE T.....Heyburn Bldg., OALR

NEWPORT

THOMASSON, W. J.....942 York St., ALR

PADUCAH

REYNOLDS, H. G.....City National Bank Bldg., OALR

RICHMOND

DUNN, MURISON.....OALR

LOUISIANA

NEW ORLEANS

BAHN, CHAS. A.....1707 Pere Marquette Bldg., Op.
BLUM, HENRY N.....1211 Maison Blanche Bldg., OALR
COLLIER, GEO. BENJAMIN.....1317 Whitney Bldg., ALR
FUCHS, VALENTINE HENRY.....Chaille Bldg., ALR
HASPEL, M. DAVID.....603 Maison Blanche Bldg., OALR
LANDFRIED, C. J.....607 Baronne Bldg., ALR
LEJEUNE, F. E.....632 Maison Blanche Bldg., ALR
LYNCH, R. C.....Maison Blanche Bldg., ALR
SMITH, VICTOR C.....1707 Pere Marquette Bldg., Op.
TAQUINO, GEORGE J.....208 Audubon Bldg., ALR
WAGNER, W. A.....1003 Pere Marquette Bldg., ALR
WEIL, A. I.....3503 Prytania St., ALR

SHREVEPORT

LARUE, C. L.....Highland Clinic, OALR
MOISE, ALLYN B.....Medical Arts Bldg., ALR
SCALES, J. L.....Medical Arts Bldg., OALR

MAINE

AUGUSTA

TURNER, OLIVER W.....Masonic Temple, OALR

PORTLAND

BEACH, S. J.....704 Congress St., Op.
GILBERT, FRANK Y.....148 Park St., OALR
GORDON, CHAS. H.....46 Deering St., ALR
HASKELL, ALFRED W.....129 Park St., Op.
HOLT, E. E., JR.....723 Congress St., Op.
O'SULLIVAN, T. J.....10 Pine St., ALR
SPALDING, J. A.....627 Congress St., Op.A

WATERVILLE

HILL, FREDERICK T.....Professional Bldg., ALR
HILL, HOWARD F.....Professional Bldg., Op.

MARYLAND

ANNAPOLIS

ALBRIGHT, LT.-COM. P. M., M.C., U. S. N.
U. S. Naval Academy, OALR
WELCH, ROBERT S. G.....46 State St. Circle, OALR

BALTIMORE

BORDLEY, JAMES, JR.....330 N. Charles St., OALR
FRIEDENWALD, HARRY.....1212 Eutaw Pl., Op.A
FRIEDENWALD, J. S.....1212 Eutaw Pl., Op.
GOLDBACK, L. J.....1012 N. Charles St., Op.
KEMLER, JOS.....1908 Eutaw Pl., OALR
LOOPER, EDWARD ANDERSON.....104 Madison St., OALR
PETERMAN, H. E.....518 Cathedral St., OALR
SAVAGE, M. M.....1720 Eutaw Pl., OALR
TARUN, WILLIAM.....104 W. Madison St., OALR
WOODS, HIRAM.....842 Park Ave., Op.A

BEVERLY

ODENEAL, THOMAS HELM.....234 Cabot St., OALR

CUMBERLAND

JONES, E. L.....First National Bank Bldg., OALR
ROBINSON, H. T.....Medical Bldg., OALR
SHARRETT, GEO. O.....119 Bedford, OALR

MASSACHUSETTS

BOSTON

BRYANT, ALICE GERTRUDE.....502 Beacon St., ALR
CARVILL, LIZZIE MAUD.....82 Commonwealth Ave., Op.
CHENERY, W. E.....377 Commonwealth Ave., ALR
CORBETT, J. J.....520 Beacon St., OALR
DERBY, GEO. S.....5 Bay State Road, Op.
FREEDMAN, L. M.....475 Commonwealth Ave., ALR
GREENWOOD, ALLEN.....82 Commonwealth Ave., Op.
HURLEY, E. D.....419 Boylston St., Op.
INGLIS, HARRY J.....43 Bay State Rd., ALR
KLEINERT, MARGARET NOYES.....82 Commonwealth Ave., ALR
KNOWLTON, CHARLES DAVISON.....520 Commonwealth Ave., ALR
LANCASTER, W. B.....529 Commonwealth Ave., Op.
LEWIS, J. P.....543 Boylston St., ALR
MOSHER, HARRIS P.....828 Beacon St., ALR
QUINCY, J. E.....270 Commonwealth Ave., ALR
RICE, G. B.....99 Bay State Road, ALR
RICHARDS, L. G.....319 Longwood Ave., ALR
ROWLAND, W. D.....220 Clarendon St., Op.
SCHALL, L. A.....270 Commonwealth Ave., ALR
SKIRBALL, J. J.....353 Commonwealth Ave., OALR
THOMPSON, PETER H.....308 Commonwealth Ave., Op.
TINGLEY, LOUISA PAINE.....9 Massachusetts Ave., Op.
TOBEY, GEO. L., JR.....270 Commonwealth Ave., ALR
WALKER, WALDO W.....99 Bay State Road, ALR
WELLS, DAVID W.....Hotel Westminister, Op.

CLINTON

MONAHAN, J. A.....181 Chestnut St., OALR

FALL RIVER

RYDER, D. R.....151 Rock St., ALR

GARDNER

BONE, HERMAN D.....19 Pleasant St., OALR

HOLYOKE

BURKE, JOHN R.....207 Elm St., OALR
HUSSEY, E. J.....245 Maple St., OALR

LAWRENCE

CONLON, F. A.....301 Essex St., OALR
MERRILL, W. HOWE.....Bay State Bldg., OALR

LOWELL

WIGGIN, WM. IRVING.....202 Central St., ALR

LYNN

JONES, E. W.....44 Atlanta St., Op.A
O'REILLY, W. F.....44 S. Common St., OALR

NEW BEDFORD

POTTER, L. F.....68 Walnut St., OALR

NORTHAMPTON

COLLINS, J. D.....187 Main St., ALR
DOW, FRANK E.....4 West St., Op.

PALMER

MOORE, G. A.....353 Main St., OALR

PITTSFIELD

THOMPSON, G. H.....74 North St., OALR

SALEM

HENNESSEY, W. W.....333 Essex St., OALR

SPRINGFIELD

BYRNES, H. F.....67 Chestnut St., OALR
CARLETON, RALPH.....10 Temple St., Op.
GALLAGHER, J. H. C.....1537 Main St., ALR
IRWIN, VINCENT J., JR.....1634 Main St., OALR
IRWIN, V. J., SR.....1634 Main St., OALR

TAUNTON

BAKER, HARRY B.....59 Main St., OALR

WORCESTER

CAHILL, J. W.....390 Main St., OALR
CROSS, A. E.....390 Main St., Op.
ESTABROOK, CHAS. T.....857 Pleasant St., OALR
MESSIER, A. E.....390 Main St., OALR
RICE, JOHN E.....390 Main St., OALR

MICHIGAN

ANN ARBOR

FURSTENBERG, A. C.....1st Nat'l Bank Bldg., ALR
PARKER, WALTER R.....1st Nat'l Bank Bldg., Op.
SLOCUM, GEO.....1st Nat'l Bank Bldg., Op.

BATTLE CREEK

COLVER, B. N.....Sanitarium 1, ALR
FRASER, R. H.....Battle Creek Sanitarium, ALR
HAUGHEY, WILFRED H.....303 Post Bldg., OALR
SLEIGHT, R. D.....303 Post Bldg., Op.
STEGMAN, LOUIE V.....Sanitarium, Op.
WENCKE, CARL G.....Battle Creek Sanitarium, ALR

BAY CITY

BAKER, CHAS. H.....Bay City Bank Bldg., OALR

DETROIT

AMBERG, EMIL.....622 Maccabee Bldg., ALR
BARNETT, SAUL E.....25 Parsons St., ALR
BEATTIE, ROBERT.....David Whitney Bldg., OALR
BEGLE, HOWELL L.....2730 East Jefferson Ave., Op.
BENTLEY, NEIL I.....1161 David Whitney Bldg., OALR
BERNSTEIN, E. J.....10 Peterboro, OALR
BROWN, A. O.....742 Maccabee Bldg., OALR
CAMPBELL, D. A.....David Whitney Bldg., OALR
CAMPBELL, DON M.....David Whitney Bldg., OALR
CAMPBELL, M. D.....David Whitney Bldg., OALR
CARTER, JOHN M.....David Whitney Bldg., ALR
CLIFFORD, T. P.....David Whitney Bldg., OALR
CONNOR, RAY.....1410 Stroh Bldg., OALR
DEFNET, WM. A.....1309 Stroh Bldg., ALR
FOWLER, WM.....1048 Maccabee Bldg., OALR
GLOWACKI, BEN FRANCIS.....Maccabee Bldg., OALR
GOUX, RAYMOND SALOT.....545 David Whitney Bldg., OALR
GRANT, LEE E.....David Whitney Bldg., OALR
HALE, ARTHUR STANLEY.....1609 Eaton Tower, OALR
HARRELL, VOSS.....Hofman Bldg., ALR
HEATH, PARKER.....David Whitney Bldg., Op.
HUGHES, RAY W.....1501 David Whitney Bldg., OALR
KEATING, T. F.....1042 Maccabees Bldg., OALR
MCCLELLAND, CARL C.....1857 David Whitney Bldg., OALR
MAIRE, LEWIS E.....710 Kresge Bldg., OALR
MERCER, R. E.....David Whitney Bldg., LR
MERKEL, CHARLES CARSON.....1409 David Whitney Bldg., ALR
MINER, S. G.....2641 E. Jefferson Ave., ALR
MORTON, J. B.....90 E. Warren Ave., OALR
PEIRCE, HORACE W.....704 Medical Arts Bldg., ALR
POOS, E. E.....Fisher Bldg., OALR
RENAUD, G. L.....510 Joseph Campan Ave., OALR
RYERSON, F. L.....Fine Arts Bldg., OALR

SANDERSON, H. H.....David Whitney Bldg., OALR
SHURLY, BURT R.....62 Adams Ave., W., ALR
SIMPSON, H. LEE.....1409 David Whitney Bldg., ALR
SISSON, RAYMOND J.....753 David Whitney Bldg., Op.
SUMMERS, WM. S.....1613 David Whitney Bldg., OALR
SUTHERLAND, JACOB MARION.....General Motors Bldg., ALR
WALKER, CHAS. C.....604 Maccabee Bldg., Op.
WENDEL, J. S.....744 David Whitney Bldg., ALR
WILLSON, W. W.....2204 Eaton Tower, OALR
WOODWORTH, WM. P.....1424 Maccabee Bldg., OALR

ESCANABA

BOYCE, W. B.....1107 Ludington St., OALR

GRAND RAPIDS

DEAN, ALFRED.....308 Medical Arts Bldg., Op.
GRANT, P. T.....1407 Grand Rapids Natl. Bk. Bldg., OALR
ROBERTSON, F. D.....Ashton Bldg., OALR
ROLLER, LOUIS A.....446 Paris Ave., S. E., Retired
SNAPP, CARL F.....Medical Arts Bldg., ALR

IONIA

SEDGWICK, O. W.....OALR

IRON MOUNTAIN

BOYCE, G. H.....1st Natl. Bk. Bldg., OALR

JACKSON

MCGARVEY, WM. EDWARD.....Jackson City Bank, ALR
WINTER, GEORGE E.....409 McBride, OALR

KALAMAZOO

FULKERSON, C. B.....308 Kalamazoo National Bank Bldg., OALR
GRANT, FRED E.....Kalamazoo National Bank Bldg., OALR
WILBUR, EDWARD P...1408 Kalamazoo Trust & Sav. Bk. Bldg., OALR

LANSING

BURDICK, AUSTIN F.....416 Tussing Bldg., OALR
ELLIS, CHARLES WARD.....Bauch Bldg., ALR

MENOMINEE

ELWOOD CALVIN R.....Spies Bldg., OALR
JONES, WM. S.....522 Sheridan Rd., OALR

MT. CLEMENS

CROMAN, JOS. M., JR.....Washington Apts., OALR

PORT HURON

FRASER, ROBT. C.....940 Military St., ALR

ST. JOSEPH

MCDERMOTT, JOHN J.....1821 Niles Ave., OALR

SAGINAW

MCKINNEY, A. R.....704 Second Natl. Bk. Bldg., OALR
ROGERS, A. S.....105 S. Jefferson Ave., OALR
WATSON, R. S.....Graebrier Bldg., OALR

TRAVERSE CITY

HOLDSWORTH, FRANK.....Peoples Savings Bk. Bldg., OALR

MINNESOTA

ALBERT LEA

FOLKEN, FRANK GARRETT.....306 Albert Lea State Bk. Bldg., OALR

BRECKENRIDGE

RICE, CLARENCE PRENTICE.....OALR

DULUTH

KNAPP, F. N.....Bradley Bldg., OALR
SHASTID, T. H.....10th Ave. Med. Bldg., Op.
TILDERQUIST, D. L.....425 Bradley Bldg., OALR
WINTER, JOHN A.....205 W. 2nd St., OALR

FERGUS FALLS

KITTELSON, THEODORE.....OALR

HIBBING

MORSMAN, L. W.....Morsman Clinic, Op.
RUBY, F. M.....1940 E. 3rd Ave., OALR
SINAMARK, ANDREW.....205 Power Bldg., OALR

MANKATO

JAMES, J. H.....National City Bank Bldg., OALR

MINNEAPOLIS

BROWN, EDWARD J.....900 Nicollet Ave., OALR
CAMP, WALTER E.....Medical Arts Bldg., OALR
CAMPBELL, ROBERT A.....520 La Salle Bldg., OALR
CLARK, H. S.....Medical Arts Bldg., OALR
DORGE, RICHARD IRVING.....2319 6th St., S., OALR
FINK, WALTER H.....426 La Salle Bldg., OALR
HANSEN, ERLING W.....910 Donaldson Bldg., OALR
HOWARD, W. H.....Medical Arts Bldg., OALR
LITCHFIELD, J. T.....609 Yeates Bldg., OALR
LOOMIS, E. A.....Medical Arts Bldg., OALR
MACNIE, J. S.....74 S. 11th St., OALR
MATTHEWS, JUSTUS.....1021 Metropolitan Bldg., LR
MORTON, HOWARD MCI.....1111 Nicollett Ave., Op.
NEWHART, HORACE.....Medical Arts Bldg., ALR
OBERG, C. M.....Syndicate Bldg., OALR
PATTERSON, W. E.....74 S. 11th St., OALR
PHELPS, KENNETH A.....730 La Salle Bldg., OALR
PRATT, FRED J.....Medical Arts Bldg., OALR
PRATT, JOHN A.....Medical Arts Bldg., OALR
REYNOLDS, J. S.....Medical Arts Bldg., OALR
RUCKER, CHAS. W.....Donaldson Bldg., Op.
SCHWARTZ, VIRGIL JOSEPH.....Medical Arts Bldg., ALR
SMITH, ARTHUR E.....Donaldson Bldg., OALR
STROUT, E. S.....74 S. 11th St., OALR

STROUT, G. E.....1009 Nicollet Ave., OALR
WATSON, JAMES A.....74 S. 11th St., OALR
WOOD, DOUGLAS P.....Medical Arts Bldg., OALR
WRIGHT, C. DARCY.....307 Yeates Bldg., OALR

NEW ULM

REINEKE, GEO. F.....Box 137, OALR

ROCHESTER

ANDERSON, CARL M.....Mayo Clinic, ALR
BENEDICT, WILLIAM L.....Mayo Clinic, Op.
FIGI, F. A.....Mayo Clinic, LR
HEMPSTEAD, BERT E.....Mayo Clinic, ALR
LILLIE, HAROLD I.....Mayo Clinic, ALR
NEW, GORDON B.....Mayo Clinic, L
PRANGEN, AVERY DE H.....Mayo Clinic, Op.
STARK, WM. B.....Mayo Clinic., ALR
WAGENER, HENRY P.....Mayo Clinic, Op.

ST. PAUL

BERRISFORD, PAUL D.....Lowry Bldg., OALR
BINGER, H. E.....Lowry Medical Bldg., OALR
BOECKMANN, EGIL.....Medical Arts Bldg., OALR
BRAY, E. R.....342 Lowry Bldg., OALR
BROWN, JOHN C.....Medical Arts Bldg., OALR
BURCH, FRANK E.....754 Linwood Pl., Op.
CONNOR, CHAS. E.....Hamm Bldg., ALR
FOGARTY, CHAS. W...St. Paul Clinic Bldg., St. Peter & 5th Sts., OALR
FULTON, JOHN F.....833 Lowry Bldg., OALR
LARSEN, CARL L.....1027 Lowry Medical Bldg., OALR
LEWIS, WILLIAM W.....836 Lowry Bldg., OALR
MALONEY, THOMAS J.....1032 Lowry Bldg., OALR
NELSON, LOUIS A.....830 Lowry Bldg., Op.
ROTHSCHILD, HAROLD J.....239 Lowry Bldg., OALR
SHELLMAN, J. L.....Lowry Medical Arts Bldg., OALR
WHEELER, MERRITT W.....1027 Lowry Bldg., OALR
WOLD, K. C.....Lowry Medical Arts Bldg., OALR

WINONA

RISSER, EDWARD D.....310 Choate Bldg., OALR
WALKER, GEO. H.....172 Main St., OALR

MISSISSIPPI

GREENVILLE

MONTGOMERY, DANIEL C.....Greenley Bldg., ALR

GULFPORT

MCWILLIAMS, CHAS. A.....G. & S. I. Bldg., ALR

JACKSON

ADKINS, GEO. E.....Jackson Infirmary, ALR
HARRIS, WM. ROBIN.....Lamar Life Bldg., ALR

NATCHEZ

GAUDET, LUCIAN S.....405 Franklin St., OALR

OXFORD

GUYTON, B. S.....OALR

VICKSBURG

BELL, M. H.....First National Bank Bldg., Op.
EDWARDS, C. J.....First National Bank Bldg., OALR
HOWARD, E. F.....First National Bank Bldg., ALR
JONES, EDLEY H.....First National Bank Bldg., OALR

MISSOURI

CARTHAGE

POST, WINFRED B.....First National Bank Bldg., OALR
POWERS, EVERETT.....Central National Bank Bldg., OALR

COLUMBIA

SNEED, CARL M.....506 W. Broadway, OALR

KANSAS CITY

ALLEN, CHAS. E.....712 Waldheim Bldg., OALR
ALTRINGER, A. N.....710 Rialto Bldg., ALR
BEAL, HOMER, A.....1002 Argyle Bldg., ALR
BEIL, J. W.....Argyle Bldg., OALR
BELLOWS, GEORGE E.....Rialto Bldg., Op.
BLAKESLEY, T. S.....Lathrop Bldg., OALR
CURDY, R. J.....1134 Rialto Bldg., Op.
CURRAN, E. J.....1805 Fed. Res. Bk. Bldg., Op.
DAVIS, HARRY B.....712 Bryant Bldg., OALR
DIXON, OTTO J.....902 Med. Arts Bldg., ALR
EUBANK, AMBROSE E.....636 Argyle Bldg., OALR
FOSTER, HAL.....1014 Chambers Bldg., ALR
HEDRICK, HAROLD B.....910 Rialto Bldg., ALR
HOWARD, J. W.....Rialto Bldg., OALR
KEITH, W. E.....Waldheim Bldg., OALR
LANING, J. H.....907 Rialto Bldg., ALR
LEA, JOS. A.....1125 Rialto Bldg., ALR
LEMOINE, A. N.....1118 Rialto Bldg., Op.
LICHTENBERG, J. S.....Rialto Bldg., Op.
LORIE, ALVIN.....Commerce Bldg., ALR
LUX, PAUL.....409 Commerce Bldg., ALR
MCALESTER, ANDREW W., JR.....625 Bryant Bldg., Op.
MCCARTY, VIRGIL W.....800 Rialto Bldg., OALR
MCKEE, JOSEPH W.....1222 Rialto Bldg., Op.
MILLER, HUGH.....Chambers Bldg., OALR
MOSS, HARVEY E.....1321 Rialto Bldg., OALR
MOTT, JOHN S.....214 Argyle Bldg., OALR
MYERS, JOHN L.....815 Shukert Bldg., OALR
PAINTER, A. M.....418 Bryant Bldg., ALR
ROBERTS, S. E.....1810 Federal Reserve Bank Bldg., ALR

SCHUTZ, W. H.....Bryant Bldg., OALR
SHOEMAKER, FERDINAND
U. S. Vet. Hosp., No. 67, 27th and Paseo Sts., OALR
SHUMATE, D. L.....500 Argyle Bldg., OALR
THOMASON, H. E.....Rialto Bldg., ALR
TEALL, RAYMOND E.....1100 Rialto Bldg., ALR
YAZEL, HERMAN E.....733 Rialto Bldg., OALR

ST. JOSEPH

FORGRAVE, L. ROBERT.....204 Logan Bldg., OALR
KENNEY, WM. L.....Corby Forsee Bldg., OALR
MINTON, WM. H.....1st State Bk. Bldg., OALR
PROUD, WILLARD C.....Phys. and Surg. Bldg., OALR

ST. LOUIS

ALDEN, ARTHUR M.....537 Frisco Bldg., ALR
ALVIS, BENNETT Y.....900 Carleton Bldg., Op.
ARBUCKLE, MILLARD F.....1011 Beaumont Med. Bldg., ALR
BARDENHEIER, F. G. A.....Frisco Bldg., Op.A
BLACK, W. D.....Metropolitan Bldg., ALR
BROWN, CLYDE O.....201 Humboldt Bldg., ALR
BURNS, S. S.....Missouri Theatre Bldg., ALR
CALHOUN, JAMES GRAHAM.....Metropolitan Bldg., Op.
CHARLES, JOS. W.....3720 Washington Ave., Op.
COSTEN, J. B.....Beaumont Medical Bldg., ALR
DAVIS, CHARLES L.....3720 Washington Blvd., ALR
DEAN, L. W.....Medical School, Washington Univ., ALR
DONNELL, N. R.....Metropolitan Bldg., Op.
DYER, C. P.....408 Humboldt Bldg., Op.
EBER, CARL THEODORE.....1006 Carleton Bldg., Op.
EIMER, CHARLES EUGENE.....316 Lister Bldg., ALR
FERRIS, DAVID P.....1000 Carelton Bldg., ALR
FREIMUTH, L. E.....234 University Club Bldg., ALR
GLICK, HARRY N.....3115 S. Grand Ave., ALR
GOLDSTEIN, M. A.....3858 Westminster Pl., ALR
GREEN, JOHN.....3720 Washington Blvd., Op.
GUGGENHEIM, L. K.....Carleton Bldg., ALR
GUNDELACH, C. A.....825 University Club Bldg., ALR
HANSEL, FRENCH KELLER.....Beaumont Medical Bldg., ALR
HARDESTY, J. F.....706 Missouri Bldg., Op.
HARDY, W. F.....Humboldt Bldg., Op.
HOBART, CARL.....Metropolitan Bldg., Op.
HOURN, GEORGE E.....511 University Club Bldg., ALR
HOWARD, HARVEY.....School of Med., Washington Univ., Op.
JACOBS, MAX W.....Missouri Theatre Bldg., Op.
JONES, VINCENT L.....3511 Washington Ave., Op.
KELLEY, ISAAC D., JR.....811 Beaumont Med. Bldg., ALR
LAMB, HARVEY D.....826 Metropolitan Bldg., Op.
LEAVY, C. A.....Metropolitan Bldg., ALR
LUEDDE, W. H.....Metropolitan Bldg., Op.

LYMAN, HARRY W.....Carleton Bldg., ALR
McMAHON, BERNARD JOHN.....Missouri Theatre Bldg., ALR
MASON, ROY EARL.....Frisco Bldg., Op.
MILLER, VERNON B.....810 Metropolitan Bldg., ALR
MYERS, EUGENE LEE.....Wall Bldg., ALR
NEMOURS, PAUL ROLAND.....3852 Greer Ave., ALR
PAYNE, R. J.....University Club Bldg., ALR
PELZ, MORT D.....Missouri Bldg., ALR
PFINGSTEN, C. F.....6370 Waterman Ave., ALR
POOS, GROVER H.....Univ. Club Bldg., OALR
POST, LAWRENCE T.....520 Metropolitan Bldg., Op.
POST, M. HAYWARD.....520 Metropolitan Bldg., Op.
POTTER, ALLEN B.....3725 Kingshighway, ALR
PROETZ, A. W.....Beaumont Bldg., ALR
REIM, HUGO.....417 Metropolitan Bldg., Op.
RYAN, L. M.....Wall Bldg., ALR
SAUER, W. E.....Beaumont Medical Bldg., ALR
SCHOLZ, R. P.....Metropolitan Bldg., ALR
SCHWARTZ, FREDERICK O.....701 Metropolitan Bldg., Op.
SENSENEY, EUGENE T.....316 Lister Bldg., ALR
SHAHAN, W. E.....Metropolitan Bldg., Op.
SHOEMAKER, W. A.....1006 Carleton Bldg., Op.
SIMON, FREDERICK C.....914 Arcade Bldg., ALR
SMIT, WM. M.....719 Missouri Theatre Bldg., ALR
TOOKER, C. W.....Carleton Bldg., Op.
VAN METER, EUGENE R.....4473 Washington Ave., ALR
WESTLAKE, SAMUEL B.....Beaumont Med. Bldg., ALR
WIENER, MEYER.....900 Carleton Bldg., Op.
WOOD, V. VISSCHER.....Beaumont Medical Bldg., ALR
WOODRUFF, FREDERICK E.....Metropolitan Bldg., Op.

SEDALIA

LOVE, JOHN G.....Ilgenfritz Bldg., OALR
TITSWORTH, GUY.....111 W. 4th St., OALR

SPRINGFIELD

BAILEY, HAROLD.....840 Landers Bldg., OALR
COFFELT, THEODORE A.....1240 Benton Ave., OALR
KLINGNER, THOS. O.....318 Landers Bldg., OALR
SMITH, CARLIE SOUTER.....214 N. Jefferson Ave., OALR

TRENTON

AMBROSE, ELMER C.....910½ Main St., OALR

MONTANA

ANACONDA

DUNLAP, LAWRENCE G.....101 Main St., OALR

BUTTE

DONOVAN, JNO. A.....507 Phoenix Bldg., OALR
MORSE, A. W.....507 Phoenix Bldg., OALR
POTTER, PETER.....125 W. Granite St., OALR

GREAT FALLS

COULTER, CHAS. F.....First Natl. Bank Bldg., OALR

HAVRE

BRIGGS, F. W.....OALR

LEWISTOWN

PARSONS, J. G.....313 Bank Electric Bldg., OALR

ROUNDUP

LEWIS, GEORGE A.....Wall Bldg., OALR

NEBRASKA

BEATRICE

TUCKER, JAMES C.....515½ Court St., OALR

COLUMBUS

CAMPBELL, C. H.....Col. State Bk. Bldg., OALR
KOEBBE, EZRA EDWIN.....ALR

HASTINGS

MARTIN, ROY DAVID.....Foote Bldg., ALR

KEARNEY

FOX, CHAS. H.....2119 Central Ave., OALR

LINCOLN

BLACK, PAUL.....Stuart Bldg., Op.
BROOKS, EARL B.....939 Stuart Bldg., OALR
CURTIS, W. L.....702 Sharp Bldg., OALR
DAYTON, W. L.....302 Funke Bldg., OALR
FURGASON, A. P.....Richards Block, OALR
HOMPES, J. J.....Sharp Bldg., OALR
SANDERSON, DAVID D.....Stuart Bldg., OALR
THOMAS, JAMES W.....1108 Sharp Bldg., OALR
ZEMER, S. G.....950 Stuart Bldg., OALR

NORFOLK

GADBOIS, ARTHUR E.....421 Norfolk Ave., OALR
NELSON, LLOYD L.....Fleming Bldg., OALR

OMAHA

BUSHMAN, L. B.....631 City Natl. Bk. Bldg., OALR
CALLFAS, W. F.....1620 Med. Arts Bldg., ALR
DAVIS, JOHN CALVIN, JR.....425 Aquila Court, OALR
FAIRCHILD, NORA M.....Med. Arts Bldg., Op.
HANEY, WM. P.....1500 Med. Arts Bldg., OALR
KNODE, A. R.....708 Med. Arts Bldg., ALR
KULLY, B. M.....Med. Arts Bldg., ALR
LEMERE, H. B.....Med. Arts Bldg., OALR
OWEN, F. S.....Med. Arts Bldg., OALR
PATTON, J. M.....Med. Arts Bldg., Op.
POTTER, GEO. B.....Med. Arts Bldg., OALR
POTTS, J. B.....Med. Arts Bldg., ALR

ROMANEK, PHILIP L.....Med. Arts Bldg., ALR
RUBENDALL, C.....Med. Arts Bldg., OALR
SWAB, CHARLES M.....Med. Arts Bldg., Op.
UREN, C. T.....537 City National Bank Bldg., ALR
WHERRY, W. P.....1500 Med. Arts Bldg., OALR

NEVADA

RENO

FULLER, JOHN ANDRE.....Farmers & Merchants Bk. Bldg., OALR

NEW HAMPSHIRE

NASHUA

NUTTER, C. F.....16 Amherst St., Op.

NEW JERSEY

ASBURY PARK

FISHER, JAMES A.....Jersey Central Bldg., OALR
UPHAM, HELEN F.....305 Third Ave., OALR

ATLANTIC CITY

CHARLTON, C. COULTER.....124 S. Illinois Ave., ALR
MCGIVERN, C. S.....101 S. Indiana Ave., ALR

BLOOMFIELD

MOORE, JOHN D.....6 Washington St., OALR

EAST ORANGE

BUVINGER, CHARLES W.....50 Washington St., OALR

HACKENSACK

GREENFIELD, WILLIAM JOHN.....418 Main St., ALR

JERSEY CITY

PYLE, WALLACE.....15 Exchange Pl., OALR

LONG BRANCH

CAMPBELL, W. K.....96 Third Ave., OALR

NEWARK

CARDWELL, EDGAR PARMELE.....12 Central Ave., ALR
CHATTIN, J. FRANKLIN.....671 Broad St., OALR
HUGHES, L. W.....1019 Broad St., OALR
HURFF, J. W.....86 Washington St., OALR
KEIM, WILLIAM F.....25 Roseville Ave., ALR
O'CONNOR, D. F.....671 Broad St., OALR
ORTON, HENRY B.....24 Commerce St., ALR
QUINBY, W. O'G.....14 James St., OALR
SHERMAN, ELBERT S.....671 Broad St., Op.
WOOD, EARL LEROY.....33 Lincoln Park, ALR
ZEHNDER, A. CHARLES.....188 Roseville Ave., Op.

ORANGE

EMERSON, LINN.....Metropolitan Bldg., OALR

PLAINFIELD

CHILDERS, ROBERT JEFFERSON.....604 Park Ave., ALR

TRENTON

ADAMS, C. F.....34 W. State St., OALR
CRANE, J. WELLINGTON.....128 Third Ave., OALR

UNION CITY

SACCO, A.....440 New York Ave., OALR

NEW MEXICO

ALBUQUERQUE

BREHMER, HARRISON L.....1st Natl. Bk. Bldg., OALR

NEW YORK

ALBANY

BEDELL, ARTHUR J.....344 State St., Op.
DOWLING, J. IVIMEY.....116 Washington Ave., OALR
HINMAN, E. E.....146 Washington Ave., ALR
JUDGE, HARRY V.....46 Willett S., Op.

BINGHAMTON

KINNE, SANFORD HALSEY.....73 Main St., ALR
RANDALL, HIRAM.....81 Main St., ALR
WATSON, H. D.....151 Front St., OALR

BROOKLYN

ARROWSMITH, HUBERT.....11 Schermerhorn St., ALR
EVANS, J. N.....23 Schermerhorn St., Op.
GOLDING, JOSEPH E.....170 Hancock St., Op.
GREENFIELD, SAMUEL D.....169 New York Ave., ALR
HANCOCK, J. C.....135 Cambridge Pl., Op.
HARGITT, CHARLES A.....17 Schermerhorn St., Op.
KRIMSKY, JOSEPH.....263 Eastern Parkway, OALR
KRUSKAL, I. D.....863 Prospect Pl., Op.
LLOYD, RALPH I.....14 Eighth Ave., Op.
LOMBARDO, MELCHIORE.....142 Joralemon St., Op.
MENGER, LOUIS CHARLES.....202 Weirfield St., ALR
MYERSON, M. C.....198 Lincoln Place, ALR
ROGERS, ROBERT M.....680 St. Johns Pl., Op.

BUFFALO

ANDREWS, H. D.....33 Allen St., Op.
BENNETT, ARTHUR G.....150 N. Pearl St., Op.
BEYER, LOUIS J.....449 Delaware Ave., ALR
BLAAUW, E. E.....190 Ashland Ave., Op.
BOZER, HERRMANN EUGENE.....468 Delaware Ave., ALR
BROWN, C. M.....510 Delaware Ave., ALR
COTT, C. C.....483 Delaware Ave., ALR
COWPER, H. W.....543 Franklin St., Op.
EDSON, R. A.....498 Delaware Ave., Op.

FAIRBAIRN, JOHN F.....503 Delaware Ave., ALR
FLAGG, J. D.....473 Virginia St., Op.A
GLOSSER, HERBERT H.....448 Franklin St., Op.
HEALY, JOHN F.....503 Delaware Ave., ALR
KOENIG, IVAN J.....40 North St., Op.
LE WIN, THURBER.....112 Linwood Ave., Op.
LEWIS, F. PARK.....454 Franklin St., Op.
MCKEE, OTTO S.....83 Bryant St., ALR
MARCH, CLARA A.....465 Ashland Ave., Op.
PHILLIPS, WM. LINTON.....131 Linwood Ave., Op.
RENNER, W. S.....341 Linwood Ave., ALR
WEED, HARRY M.....196 Linwood Ave., Op.
WURTZ, W. J. M.....333 Linwood Ave., ALR

CORTLAND

HIGGINS, R. P.....20 Court St., OALR

DUNKIRK

JACKLE, ARTHUR F.....63 E. 4th St., Op.

FLUSHING

GOODE, EDWARD ALLEN.....239 Sanford Ave., ALR

GENEVA

SPENGLER, JOHN ARTHUR.....423 S. Main St., Op.

GLENS FALLS

FIELDING, F. G.....70 Ridge St., ALR

JAMESTOWN

REGER, H. S.....Physicians Bldg., OALR

LOCKPORT

RINGUEBERG, E. N. S.....13 Main St., Op.

MT. VERNON

KEEFE, WM. JOHN .....110 E. Sidney Ave., ALR
THOMSON, J. J.....531 E. Lincoln Ave., ALR

NEW ROCHELLE

BECK, A. L.....421 Huguenot St., OALR
WOODRUFF, WALTER STUART.....421 Huguenot St., ALR

NEW YORK CITY

ALMOUR, RALPH W.....51 W. 73rd St., ALR
AUERBACH, JULIUS.....1016 Fifth Ave., ALR
BALLIN, M. J.....20 E. 76th St., ALR
BEACH, B. S.....140 W. 86th St., Op.
BERENS, CONRAD.....30 E. 40th St., Op.
BRUDER, JOSEPH.....57 W. 57th St., OALR
CARTER, W. W.....2 E. 54th St., ALR
COHEN, MARTIN.....1 W. 85th St., Op.
COHN, FELIX.....47 W. 87th St., ALR
COLBERT, ROBERT MARTIN.....160 W. 87th St., ALR
COTT, D. N.....141 St. Marks Pl., St. George, Staten Isl., ALR

CUNNING, DANIEL S.....114 E. 54th St., OALR
DAVIDSON, MORRIS.....210 W. 70th St., Op.
DAVIS, A. EDWARD.....40 E. 61st St., Op.
DAVIS, G. E. .....1060 Fifth Ave., OALR
DIXON, G. S.....218 Second Ave., R
DLUGASCH, LOUIS.....110 E. 36th St., ALR
DOHERTY, W. B.....150 W. 55th St., Op.
DUNNINGTON, J. H.....30 W. 59th St., Op.
ELLIS, Z. H.....30 W. 59th St., Op.
FERNLUND, GEO. BYRON
Hillside Ave. and 118th St., Richmond Hill, OALR
FOWLER, ROBERT HENRY.....153 E. 62nd St., ALR
FOWLKES, JOHN WINSTON.....30 W. 59th St., ALR
FRANKEL, EDWARD, JR.....152 W. 58th St., ALR
FREUDENTHAL, WOLFF.....24 W. 88th St., ALR
GATEWOOD, W. L.....330 Park Ave., ALR
GOLDMAN, A. MILTON.....121 E. 60th St., Op.
GOLDMAN, H. G.....57 W. 58th St., ALR
GOLDSTEIN, ISADORE.....1060 Park Ave., Op.
GOTTLIEB, MARK J.....2 W. 87th St., ALR
GRESSER, EDWARD BELLAMY.....39 Fifth Ave., Op.
GRUSHLOW, ISRAEL.....271 Central Park, W., ALR
GUTTMAN, J.....987 Madison Ave., OALR
HALLETT, G. DEWAYNE.....274 W. 86th St., Op.
HARDY, LEGRAND H.....30 E. 40th St., Op.
HARRIS, THOMAS JEFFERSON.....104 E. 40th St., ALR
HARTSHORNE, ISAAC.....30 W. 59th St., Op.
HASKIN, W. H.....57 W. 57th St., ALR
HAYS, HAROLD M.....64 E. 58th St., ALR
HETRICK, L. E.....40 E. 61st St., ALR
HORN, WALTER L.....1040 Park Ave., ALR
HURD, L. M.....39 E. 50th St., ALR
HUGHES, WENDELL LOCHEAD.....20 E. 53rd St., Op.
HUNT, WESTLEY MARSHALL.....33 E. 68th St., ALR
HUTCHINSON, ALBERT T.....30 W. 59th St. ALR
IMPERATORI, C. J.....17 E. 38th St., ALR
INGERMAN, S. M.....418 Central Park, W., OALR
IRWIN, FRANK N.....114 E. 54th St., Op.
JACOBS, S. M.....1018 E. 163rd St., OALR
JARECKY, HERMAN.....168 W. 86th St., OALR
JOHNSON, THOMAS HOLLAND.....30 W. 59th St., Op.
JOSEPHSON, EMANUEL M.....993 Park Ave., ALR
JUDD, H. B.....40 E. 61st St., ALR
KELLY, JOSEPH DOMINIC.....114 E. 54th St., OALR
KEY, BEN WITT.....100 W. 59th St., Op.
KING, J. J.....30 E. 40th St., ALR
KIRBY, D. B.....30 W. 59th St., Op.
KNAPP, ARNOLD.....10 E. 54th St., Op.
KOPETZKY, S. J.....51 W. 73rd St., ALR

KRUG, ERNEST, F.....12 W. 44th St., Op.
LA VIGNE, A. A.....471 Park Ave., OALR
LUBMAN, MAX.....17 E. 96th St., ALR
MCCOY, JOHN.....730 Fifth Ave., ALR
MCDANNALD, C. E.....100 W. 59th St., Op.
MCDONALD, DENNIS J.....17 E. 38th St., ALR
MAY, CHAS. H.....698 Madison Ave., Op.
MAYBAUM, J. L.....1079 Park Ave., ALR
MAYER, EMIL.....666 West End Ave., ALR
MEIERHOF, E. L.....12 E. 86th St., OALR
MILLER, JOSEPH WILLIAM.....305 E. 17th St., ALR
MINSKY, HENRY.....215 W. 88th St., Op.
MITTENDORF, A. D.....339 Park Ave., Op.
MITTENDORF, W. KEITH.....115 E. 53rd St., Op.
MORRISON, WM. WALLACE.....39 E. 50th St., ALR
NISSELSON, MAX.....754 Beck St., ALR
NORTHCUTT, THOMAS A.....17 E. 38th St., Op.
OAST, SAMUEL P.....30 E. 40th St., Op.
PALMER, ARTHUR.....667 Madison Ave., ALR
PETERSON, R. A.....150 Fifth Ave., OALR
PHILLIPS, W. C.....30 E. 58th St., ALR
REESE, ALGERNON B.....50 W. 52nd St., Op.
ROSENBAUM, MORRIS.....61 W. 88th St., Op.
SAMUELS, BERNARD.....57 W. 57th St., Op.
SCAL, JOSEPH COLEMAN.....44 W. 74th St., ALR
SCHILLER, A. N.....249 West End Ave., ALR
SCHLIVEK, KAUFMAN.....1016 Fifth Ave., Op.
SCHWARTZ, A. A.....941 Park Ave., ALR
SOMBERG, J. S.....60 E. 67th St., Op.
STARK, JESSE B.....45 Park Ave., Op.
STURGES, LEIGH F.....121 E. 60th St., ALR
WEEKS, WEBB WILLIAM.....20 E. 53rd St., Op.
WHEELER, J. M.....30 W. 59th St., Op.
WIENER, ALFRED.....550 Park Ave., OALR
YANKAUER, SIDNEY.....121 E. 60th St., ALR

NIAGARA FALLS

PRICE, NORMAN W.....445 Third St., OALR
ROOKER, A. M.....225 Gluck Bldg., ALR

NYACK

TOMS, S. W. S.....120 S. Broadway, OALR

OLEAN

TINDOLPH, L. W.....First Natl. Bank Bldg., ALR

ONEIDA

CROCKETT, R. L.....302 Main St., Op.

PLATTSBURG

SCHNEIDER, ANTON S.....56 Court St., Op.A.

POUGHKEEPSIE

KRIEGER, WM. A.....35 Market St., OALR

ROCHESTER

BARBER, F.....75 S. Fitzhugh St., Op.
BARLOW, ROY A.....Hiram-Sibley Bldg., ALR
CARROLL, GEO. G.....5 North Goodman St., OALR
CLARK, L. H.....337 Monroe Ave., OALR
GIPNER, J. F.....53 S. Fitzhugh St., Op.
JONES, L. W.....16 N. Goodman St., OALR
KENNEDY, ELDRED WESTON.....Gas & Electric Bldg., Op.
LERNER, MACY LEVI.....332 Park Ave., Op.
MCDOWELL, N. D.....275 Alexander St., OALR
MORRIS, A. G.....303 Alexander St., ALR
NASH, C. STEWART.....26 S. Goodman St., ALR
SHAPERO, I. M.....289 Alexander St., OALR
SNELL, ALBERT C.....53 S. Fitzhugh St., Op.

SYRACUSE

BRITTEN, G. S.....Medical Arts Bldg., ALR
BROWN, MORTIMER G.....Medical Arts Bldg., ALR
BRUST, H. O.....820 S. Crouse Ave., ALR
CAVE, LINUS S.....221 Physicians Bldg., ALR
FOWLER, S. R.....University Block, ALR
GILLETTE, D. F.....State Tower Bldg., Op.
HALL, EDWIN P.....State Tower Bldg., ALR
HOOPLE, GORDON D.....Medical Arts Bldg., ALR
JOY, HAROLD HENRY.....504 State Tower Bldg., Op.
KAUFFMAN, A. B.....Chimes Bldg., ALR
KLINE, H. G.....State Tower Bldg., ALR
MARLOW, SEARLE B.....1004 State Tower Bldg., Op.
MOORE, ROY S.....713 E. Genesee St., ALR

TROY

SULZMAN, FRANK MALCOLM.....1831 Fifth Ave., OALR

UTICA

BEATTIE, WILLIAM H.....258 Genesee St., OALR

WATERTOWN

ATKINSON, WALTER S.....168 Sterling St., Op.

WHITE PLAINS

MORIARTY, RICHARD W.....57 Maple Ave., OALR

WOODHAVEN

FROTHINGHAM, EDWARD.....8644 94th St., OALR

NORTH CAROLINA

ASHEVILLE

BRIGGS, H. H.....101 Haywood St., OALR
GREENE, JOSEPH BERRY.....305 Haywood Bldg., ALR
MCCLELLAN, W. E.....101 Haywood St., OALR

CHARLOTTE

MATHESON, J. P.....6 W. 7th St., ALR
PEELER, C. N.....6 W. 7th St., OALR
SLOAN, HENRY L.....6 W. 7th St., Op.
WHISNANT, A. M.....517 Professional Bldg., OALR

GREENSBORO

PEERY, VANCE PRICE.....ALR

KINSTON

REAVES, CHAS. R.....Reaves Infirmary, 117 W. Sycamore St., OALR
REAVES, W. P.....Reaves Infirmary, 117 W. Sycamore St., OALR

MOREHEAD CITY

JOHNSON, OTIS H.....OALR

NEW BERN

DANIELS, R. L.....Elks Bldg., OALR

RALEIGH

HICKS, VONNIE MONROE.....Professional Bldg., Op.
WRIGHT, J. B.....Professional Bldg., ALR

WILMINGTON

MURPHY, J. G.....Murchison Bldg., OALR

WINSTON-SALEM

DAVIS, THOMAS W.....210 O'Hanlon Bldg., OALR

NORTH DAKOTA

BISMARCK

CONSTANS, GEORGE MAURICE.....Quain & Ramstad Clinic, Op.

FARGO

RINDLAUB, ELIZABETH P.....620 Front St., OALR

MINOT

MCCANNEL, A. D.....33 Central Ave., W., OALR

OHIO

AKRON

BROWN, L. E.....2nd Natl. Bank Bldg., OALR
MOORE, T. K.....416 Central Sav. & Trust Bldg., OALR
STEVENSON, D. W.....165 E. Market St., OALR

ALLIANCE

KING, GEORGE L., JR.....537 Market St., Op.
KING, G. L.....537 E. Market St., OALR

BELLEFONTAINE

BUTLER, ROBERT H.....112 W. Columbus Ave., OALR
HARBERT, JOHN P.....135 N. Main St., OALR

CANTON

HOFFMAN, JOHN N.....309 Brant Bldg., ALR
SCHILD, E. H.....Brant Bldg., Op.

CINCINNATI

ABRAHAMSON, IRA A.....605 Traction Bldg., Op.
ASBURY, MARY K.....3549 Holly Lane, Op.
BEYER, A. G.....19 Garfield Pl., OALR
BROWN, ALBERT LOUIS.....2600 Union Central Bldg., Op.
GOODYEAR, H. M.....556 Doctors Bldg., ALR
HANS, C. L.....19 W. 7th St., OALR
HARRIS, W. C.....Groton Bldg., ALR
HOFLING, CHARLES A.....707 Race St., Op.
IGLAUER, SAMUEL.....707 Race St., ALR
JONES, C. C.....656 Doctors Bldg.,ALR
KING, CLARENCE.....Union Central Bldg., Op.
KING, ED.....707 Race St., ALR
LYLE, DONALD J.....19 W. 7th St., OALR
MITHOEFER, W.....19 Garfield Pl., ALR
MURPHY, W. E.....707 Race St., OALR
PHINNEY, F. D.....22 W. 7th St., OALR
RAY, VICTOR.....19 W. 7th St., Op.
SIEGEL, FRANCIS X.....901 Union Central Life Bldg., OALR
STANBERRY, HENRY.....556 Doctors Bldg., Op.
STEWART, T. M.....901 Union Trust Bldg., OALR
STITT, H. L.....656 Doctors Bldg., OALR
TANGEMAN, H. F.....606 Union Central Life Bldg., ALR
THOMPSON, E. H.....8 W. 9th St., OALR
URNER, M. H.....2600 Union Central Bldg., OALR
VAIL, DERRICK T., SR.....24 E. 8th St., Op.
VAIL, H. H.....24 E. 8th St., ALR

CLEVELAND

ABBOTT, W. J.....8314 Euclid Ave., OALR
BRUNER, ABRAM BRENEMAN.....1214 Guardian Bldg., OALR
BRUNER, WM. E.....Guardian Bldg., Op.
BURKE, T. A.....Rose Bldg., OALR
COGAN, J. E.....707 Rose Bldg., OALR
CUTLER, FRANKLIN E.....1025 Schofield Bldg., ALR
DIXON, FRED WILLIS.....1029 Rose Bldg., ALR
FORSYTHE, S. T.....600 Rose Bldg., OALR
KENDALL, M. R.....Osborn Bldg., ALR
LARGE, S. H.....Hanna Bldg., ALR
LENKER, JOHN N.....Osborne Bldg., ALR
MCDONALD, C. L.....964 Rose Bldg., OALR
METZ, ROY B.....1214 Guardian Bldg., Op.
METZENBAUM, MYRON.....Rose Bldg., ALR
MONSON, S. H.....1322 B. F. Keith Bldg., OALR
MOTTO, MICHAEL PAUL.....550 Rose Bldg., Op.
MULLIN, W. V.....Cleveland Clinic, Euclid Ave. at 93rd St., ALR
MUSSUN, W. G.....Hanna Bldg., ALR
NELSON, CHAS. F.....606 Schofield Bldg., OALR
PITKIN, C. E.....Osborn Bldg., ALR
PRENDERGAST, D. A.....Hanna Bldg., OALR

QUITTNER, S. S.....3912 Prospect Ave., OALR
RIEGER, W. H.....Hanna Bldg., ALR
ROSENBERGER, HARRY CLIFFORD.....Rose Bldg., ALR
RUEDEMANN, ALBERT DARWIN.....Cleveland Clinic, Op.
RUST, E. G.....Hanna Bldg., Op.A
SHACKLETON, W. E.....2323 Prospect Ave., Op.
SHIRAS, HOWARD H.....624 Guardian Bldg., Op.
SHIREY, O. M.....Osborn Bldg., ALR
SIMONDS, O. F.....Hanna Bldg., Op.
STOTTER, ARTHUR L.....1148 Euclid Ave., OALR
TRIPP, IRA A.....436 Rose Bldg., OALR
TUCKERMAN, W. C.....733 Osborn Bldg., Op.
TUCKERMAN, W. H.....733 Osborn Bldg., OALR
WAUGH, J. M.....Cleveland Clinic, Euclid Ave. at 93rd St., OALR

COLUMBUS

BEATTY, H. G.....681 E. Broad St., ALR
BROWN, JOHN E.....370 E. Town St., OALR
CLARK, I. G.....188 E. State St., OALR
FROST, ALBERT DALBEY.....681 E. Broad St., Op.
HAUER, A. M.....327 E. State St., ALR
PROUT, A. W.....370 E. Town St., OALR
SANOR, DANIEL G., JR.....206 E. State St., ALR
SILBERNAGEL, C. E.....240 E. State St., OALR
STOUTENBOROUGH, WM. A.....318 E. State St., OALR
SULZER, G. A.....327 E. State St., OALR
THOMAS, FRANCIS W.....327 E. State St., OALR
TIMBERMAN, ANDREW.....Beggs Bldg., OALR

DAYTON

BINKLEY, ROY S.....530 Fidelity Bldg., OALR
BLACKBURN, W. J.....663 Reibold Bldg., OALR
DUTROW, H. V.....1040 Fidelity Medical Bldg., OALR
FARMER, A. G.....Fidelity Medical Bldg., OALR
FOUTS, JOHN D.....910 Fidelity Medical Bldg., OALR
HARRIS, H. B.....Fidelity Medical Bldg., OALR
KILBOURNE, P. H.....Fidelity Medical Bldg., OALR
MILLETTE, J. W.....Reibold Bldg., OALR

ELYRIA

GILL, GEORGE.....Elyria Sav. & Trust Bldg., OALR

IRONTON

DUNN, O. B.....Fourth and Railroad Sts., OALR

LIMA

STUEBER, F. G.....209 W. North St., OALR
STUEBER, PAUL J.....209 W. North St., OALR

LORAIN

MONOSMITH, O. B.....O. P. S. Bldg., OALR

MANSFIELD

GARBER, J. M.....14 N. Mulberry St., OALR
WISE, RALPH C.....News Bldg., OALR

MARIETTA

SAUER, W. W.....231 Third St., OALR

MARION

BRICKLEY, DANIEL W.....Uhler-Phillips Bldg., OALR

MASSILLON

SCOTT, MERLE E.....1st Natl. Bk. Bldg., OALR

MIDDLETOWN

WILLIAMS, WALTON H.....Castell Bldg., OALR

NEWARK

HATCH, C. B.....3 W. Church St., OALR

SPRINGFIELD

EASTON, J. C.....1st Natl. Bk. Bldg., OALR
HARTLEY, F. A.....302 E. High St., OALR
HOGUE, D. W.....34 W. High St., OALR
MINOR, C. L.....1st Natl. Bk. Bldg., OALR

STEUBENVILLE

GOURLEY, G. F.....Sinclair Bldg., OALR

TIFFIN

PORTER, E. H.....85 Madison St., OALR

TOLEDO

ALDERDYCE, WM.....513 Madison Ave., OALR
ALTER, F. W.....316 Michigan St., OALR
DENMAN, IRA O.....330 Richardson Bldg., OALR
KING, CHAS. R.....Spitzer Bldg., OALR
LUKENS, CHARLES.....316 Michigan St., OALR
SNYDER, W. H.....220 Michigan St., OALR
STEINFELD, A. L.....125 Fifteenth St., OALR
WINGER, IRA B.....225 Michigan St., OALR

WARREN

STIMSON, GEORGE W.....Packard Bldg., OALR

XENIA

MADDEN, REED.....Allen Bldg., OALR
SHIELDS, LAWRENCE.....Allen Bldg., ALR

YOUNGSTOWN

BEARD, HAROLD JOLLY.....510 Dollar Bank Bldg., OALR
GIBSON, R. D.....310 Wick Ave., OALR
HARTZELL, S. M.....Dollar Savings & Trust Co., OALR

ZANESVILLE

CULBERTSON, L. R.....Masonic Temple, OALR

OKLAHOMA

BARTLESVILLE

KISER, J. D. ..... Cent. Natl. Bk. Bldg., OALR

ENID

PIPER, ARTHUR S. ..... 610 S. Monroe, OALR

GUTHRIE

BARKER, CHAS. B. ..... 224½ W. Okla. Ave., OALR

McALESTER

DAVIS, J. E. ..... Kress Bldg., OALR

MUSKOGEE

FULLENWIDER, C. M. ..... Barnes Bldg., OALR

OKLAHOMA CITY

DIXON, W. E. ..... 503 Medical Arts Bldg., OALR
FERGUSON, E. S. ..... 902 Medical Arts Bldg., OALR
GUTHRIE, A. L. ..... 1010 Medical Arts Bldg., OALR
McHENRY, D. D. ..... 604 Medical Arts Bldg., OALR
NEWTON, L. A. ..... 609 Medical Arts Bldg., OALR

TULSA

BRASWELL, JAMES C. ..... Med. Arts Bldg., OALR
COOK, W. ALBERT ..... Med. Arts Bldg., OALR
HARALSON, CHARLES HERNDON ..... Med. Arts Bldg., Op.
ROTH, ALBERT W. ..... Med. Arts Bldg., OALR
SMITH, RURIC N. ..... Med. Arts Bldg., ALR

OREGON

BAKER

GORDON, GLENN E. ..... ALR

LA GRANDE

BOUVY, L. B. ..... Foley Bldg., OALR

PORTLAND

AINSLIE, GEORGE ..... Sellings Bldg., OALR
BOUVY, HARRY M. ..... 800 Pittock Blk., OALR
BRUERE, GUSTAVE E. ..... Medical-Dental Bldg., OALR
CARRUTH, HOWARD ERNEST ..... 716 Med. Arts Bldg., OALR
DAVIS, RALPH F. ..... Mayer Bldg., OALR
DICKSON, JOHN F. ..... 418 Mayer Bldg., OALR
FENTON, RALPH A. ..... 806 Medical Arts Bldg., OALR
HENTON, G. E. ..... Morgan Bldg., OALR
JOHNSTON, WILSON ..... 611 Lovejoy St., OALR
KARKEET, ROBERT B. ..... 1203 Selling Bldg., OALR
KIEHLE, F. A. ..... 410 Medical Arts Bldg., OALR
KISTNER, F. B. ..... 600 Mayer Bldg., OALR
LUCAS, HAROLD R. ..... Medical-Dental Bldg., OALR
LUPTON, IRVING MARTIN ..... Medical Arts Bldg., OALR
WEEKS, JOHN E. ..... Dunthorpe, Rt. 8, (Retired), Op.

PENNSYLVANIA

ALTOONA

GLOVER, S. P.....1118 Twelfth Ave., OALR

BEAVER

CRUMRINE, NORMAN ROSS.....595 Third St., OALR
SCROGGS, J. J.....218 E. Third St , Op.A

BETHLEHEM

CHASE, W. D.....230 E. Broad St., ALR

BRADFORD

ASH, GARRETT G.....1 Main St., OALR

BUTLER

DOANE, L. L.....357 N. Main St., OALR
HAZLETT, L. R.....Savings & Trust Bldg., OALR

CHARLEROI

FERMAN, JOHN W.....Stahlman Bldg., OALR

CHESTER

CROSS, G. H. .....525 Welsh St., Op.
ORR, ADRIAN V. B.....525 Welsh St., ALR

CONNELLSVILLE

HARRISON, FRED H.....308 1st Natl. Bk. Bldg., OALR

ERIE

DENNIS, DAVID N.....221 W. 9th St., Op.
DENNIS, EDWARD P.....309 W. 10th St., OALR
FUST, JOHN H. E.....221 W. 9th St., OALR
HEARD, C. F.....813 Sassafras St., OALR
JACKSON, J. D.....232 W. 8th St., OALR
RUSSELL, J. A. M.....813 Sassafras St., OALR
SCHLINDWEIN, G. WM.....138 W. 9th St., OALR
SHREVE, OWEN M.....162 W. 8th St., Op.

FRANKLIN

JOBSON, G. B.....West Park and Liberty St., OALR

GREENSBURG

FAIRING, JOHN W.....206 Tribune Review Bldg., OALR
MCKEE, CLAUD W.....Coulter Bldg., OALR

HARRISBURG

DAILEY, GILBERT L.....618 N. 3rd St., OALR
FARNSLER, H. HERSHEY.....1438 Market St., OALR
GRACEY, GEO. F.....209 State St., OALR
PARK, J. WALTER.....32 N. 2nd St., OALR

HAZLETON

REICHE, O. C.....Markle Bk. Bldg., OALR

JOHNSTOWN

BARKER, O. G. A.....804 Johnstown Trust Bldg., Op.
HARRIS, C. M.....604 Johnstown Trust Bldg., OALR
RUSH, C. C.....907 U. S. Natl. Bk. Bldg., OALR

LEBANON

REESE, MONROE D.....123 N. 8th St., OALR

MIDDLETOWN

GEORGE, HENRY W.....19 N. Union St., Op.

NEW KENSINGTON

MCCONNELL, T. E.....Logan Trust Bldg., OALR
STEIM, J. M.....1st Natl. Bk. Bldg., Op.

NORRISTOWN

BEIDEMAN, JOSEPH ELLSWORTH...Montgomery Trust Arcade, Op.
PRICE, JOHN BEADLE.....513 Swede St., ALR

PHILADELPHIA

ADLER, FRANCIS HEED.....313 S. 17th St., Op.
ALEXANDER, GEO. J.....1629 Spruce St., ALR
AMANO, KAGEYAS W.....502 Woodland Terrace, ALR
ANNON, W. T.....235 S. 15th St., ALR
APPLEMAN, L. F.....308 S. 16th St., Op.
BABBITT, JAMES A.....1912 Spruce St., ALR
BAESEMAN, R. WINFIELD.....928 Clinton St., ALR
BALDWIN, KATE W.....1117 Spruce St., ALR
BALENTINE, P. L.....1524 Chestnut St., Op.
BLAIR, MORTIMER W.....N. E. Cor. 20th & Chestnut St., Op.
BOEHRINGER, W. H. .....700 Central Medical Bldg., OALR
BROWN, SAMUEL HORTON.....1930 Chestnut St., Op.
BRUMM, SETH A.....Medical Arts Bldg., OALR
BUTLER, MARGARET F.....1831 Chestnut St., ALR
BUTT, MIRIAM M.....2045 Chestnut St., Op.
CLERF, LOUIS HENRY.....128 S. 10th St., ALR
COATES, GEO. M.....1721 Pine St., ALR
COHEN, HERMAN B.....Spruce and 13th Sts., ALR
COHEN, SAMUEL.....2009 Pine St., ALR
COWAN, ALFRED.....1930 Chestnut St., Op.
CREIGHTON, WM. J.....258 S. 18th St., Op.
CROSKEY, J. W.....Medical Arts Bldg., Op.
DAVIS, J. L.....135 S. 18th St., ALR
DEICHLER, LYN W.....1737 Chestnut St., OALR
DINTENFASS, HENRY.....1305 Spruce St., ALR
EMBERY, FRANK.....1532 Overington St., ALR
ERSNER, MATTHEW S.....1915 Spruce St., ALR
FELT, C. L.....Chestnut and 20th Sts., ALR
FOX, L. WEBSTER.....303 S. 17th St., Op.
GLEASON, E. B.....2112 Pine St., ALR
GODDARD, H. M.....1531 Spruce St., ALR
GOWEN, FRANCIS V.....2033 Pine St., ALR
GRISCOM, J. MILTON.....2213 Walnut St., Op.
HAWMAN, ERLE G.....4051 N. Broad St., Op.
HEED, C. R.....1205 Spruce St., Op.
HOLLOWAY, THOS. B.....1819 Chestnut St., Op.
HUSIK, D. N.....1930 Chestnut St., ALR

JACKSON, CHEVALIER.....235 S. 15th St., L
KAUFMAN, A. SPENSER.....1923 Spruce St., ALR
KEELER, J. C.....Medical Arts Bldg., ALR
KRAUSS, FREDERICK.....1703 Chestnut St., OALR
LAESSLE, HENRY A.....The Lenox, 13th and Spruce Sts., ALR
LAWRENCE, GRANVILLE A.....Medical Arts Bldg., Op.
LEWIS, FIELDING O.....259 S. 17th St., ALR
LOFTUS, JOHN EDWARD.....605 Medical Arts Bldg., ALR
LUKENS, ROBERT McD.....1923 Spruce St., ALR
MACKENZIE, ALICE V.....1724 Spruce St., OALR
MACKENZIE, GEORGE W.....1724 Spruce St., OALR
MILLER, E. B.....2008 Walnut St., Op.
MITCHELL, E. K.....710 W. Lehigh Ave., ALR
MOORE, GEO. H.....18th and Chestnut Sts., OALR
MOORE, WILLIAM FREDERICK.....255 S. 16th St., ALR
OLSHO, S. L.....235 S. 15th St., OALR
O'REILLY, CHARLES A.....521 Medical Arts Bldg., ALR
PETER, L. C.....1930 Chestnut St., Op.
PONTIUS, P. J.....1831 Chestnut St., Op.
POSEY, WM. CAMPBELL.....2049 Chestnut St., Op.
RADCLIFFE, McCLUNEY.....1906 Chestnut St., Op.
REESE, WARREN S.....230 S. 21st St., Op.
RIDPATH, ROBT. F.....N. E. Cor. 18th and Chestnut Sts., LR
ROMMEL, JOHN C.....3701 N. Broad St., Op.
RUDOLPHY, JAY BESSON.....1924 Pine St., Op.
SARTAIN, PAUL J.....2006 Walnut St., Op.
SCARLETT, HUNTER W.....230 S. 21st St., Op.
SCHNEIDEMAN, T. B.....2045 Chestnut St., Op.
SCHWEINITZ, GEO. E. DE.....1705 Walnut St., Op.
SENER, WALTER J.....Physicians Bldg., Op.
SHANNON, C. E. G.....1930 Chestnut St., Op.
SHEMELEY, WM. G., JR.....1724 Spruce St., OALR
SHUSTER, BENJAMIN HARRISON.....1930 Chestnut St., ALR
SKILLERN, ROSS HALL.....1811 Spruce St., R
SKILLERN, S. R., JR.....1734 Pine St., ALR
SPAETH, EDMUND B.....1930 Chestnut St., Op.
SPENCER, W. H.....1830 S. Rittenhouse Sq., ALR
STILES, CHAS, M.....1130 Spruce St., Op.
STOUT, PHILIP S.....269 S. 19th St., ALR
STROUSE, FREDERICK M.....1301 Spruce St., ALR
TASSMAN, ISAAC S.....Medical Arts Bldg., Op.
TUCKER, GABRIEL....Graduate Hosp., 19th and Lombard Sts., B
WAGERS, ARTHUR J.....1429 Spruce St., ALR
WEINSTEIN, M. A.....1309 Spruce St., ALR
WHELAN, G. L.....2100 Walnut St., ALR
WILLIAMS, HORACE J.....5908 Green St., Germantown, ALR
ZENTMAYER, WILLIAM..Suite 803, S. E. Cor. 20th & Chestnut, Op.

PITTSBURGH

BERNATZ, C. F.....Park Bldg., Op.
BLAIR, W. W.....121 University Pl., Op.
CARSON, W. E.....Jenkins Arcade Bldg., Op.
CURRY, GLENDON E.....Westinghouse Bldg., Op.
DAY, EWING W.....121 University Pl., OALR
FISCHER, N. A.....623 Union Trust Bldg., ALR
GROSS, JULIUS E.....8030 Jenkins Arcade Bldg., ALR
GUERINOT, A. J.....604 Westinghouse Bldg., OALR
HAGEMANN, JOHN A.....Highland Bldg., ALR
HECKEL, EDWARD B.....Jenkins Arcade Bldg., Op.
JAQUISH, CHARLES J.....904 Westinghouse Bldg., ALR
KNEEDLER, G. C.....Professional Bldg., ALR
KREBS, ADOLPH.....Jenkins Arcade Bldg., Op.
LINN, J. G.....7026 Jenkins Arcade Bldg., Op.
MCCREADY, J. H.....121 University Pl., ALR
MCKEE, GEO. J.....Westinghouse Bldg., ALR
MACLACHLIN, A. A.....May Bldg., ALR
MARKEL, J. CLYDE.....Westinghouse Bldg., Op.
MARSHALL, WATSON.....121 University Pl., ALR
MEANOR, WM. C.....619 Westinghouse Bldg., Op.
PATTERSON, ELLEN J.....121 University Pl., ALR
PLUMER, JOHN S.....121 University Pl., Op.
SEEGMAN, SIMON.....3401 Fifth Ave., ALR
SHUMAN, G. H.....Park Bldg., Op.
SIMPSON, JOHN REID.....825 Highland Bldg., ALR
SMITH, STANLEY.....121 University Pl., Op.
STIEREN, E.....626 Union Trust Bldg., Op.
STURM, S. A.....7036 Jenkins Arcade Bldg., Op.
TAYLOR, M. C.....1024 May Bldg., Op.
TODD, GROVER CLEVELAND.....307 Westinghouse Bldg., ALR
TURNER, H. H.....902 Professional Bldg., Op.
VANKIRK, V. E.....626 Union Trust Bldg., Op.
WEISSER, E. A.....806 May Bldg., Op.
YOST, WALTER MILTON.....Westinghouse Bldg., Op.

READING

RHODE, SOLON L.....238 N. 6th St., OALR
STOCKLER, J. A.....332 N. 5th St., OALR

RIDGEWAY

MCALLISTER, J. C.....10 S. Mill St., AL

ROCHESTER

YOST, WALTER M.....Peoples Bk. Bldg., Op.

SAYRE

WEINBERGER, NELSON S.....Robert Packard Hosp., OALR

SCRANTON

CORSER, J. B.....345 Wyoming Ave., Op.
CURTIN, EUGENE A.....703 Connell Bldg., OALR

SULLIVAN, J. J., JR.....228 Scranton Life Bldg., ALR
WILLIAMS, M. M.....Medical Arts Bldg., Op.

SUNBURY

BECKER, H. M.....49 S. 4th St., OALR

WARREN

BALL, MICHAEL V.....316 Hazel St., OALR

WASHINGTON

MCCULLOUGH, CLARENCE JOSEPH....Washington Trust Bldg., ALR
MCKENNAN, JAS. W.....514 Washington Trust Bldg., Op.
MCMURRAY, J. B.....Washington Trust Bldg., OALR

WAYNESBURG

STAHLMAN, FRED C.....Commercial Bldg., OALR

WILKES-BARRE

BUCKMAN, E. U.....70 S. Franklin St., OALR
CARR, GEO. W.....54 S. Franklin St., OALR
KISTLER, O. F.....43 N. Franklin St., Op.AR
MCLAUGHLIN, P. A.....71 S. Washington St., OALR
SCHAPPERT, N. L.....59 S. Washington St., OALR

YORK

KLINEDINST, JOHN FERD.....220 S. George St., Op.A

RHODE ISLAND

NEWPORT

JACOBY, D. P. A.....106 Touro, Op.AL

PROVIDENCE

ADAMS, FRANK M.....122 Waterman St., ALR
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CAPRON, F. P.....118 Angell St., OALR
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